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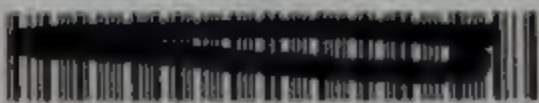
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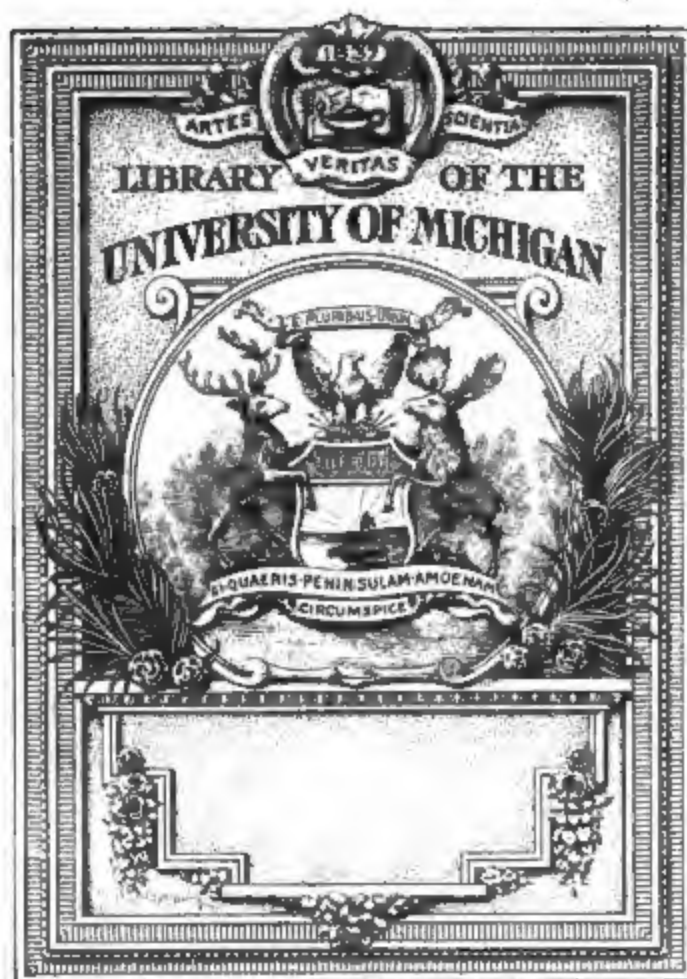
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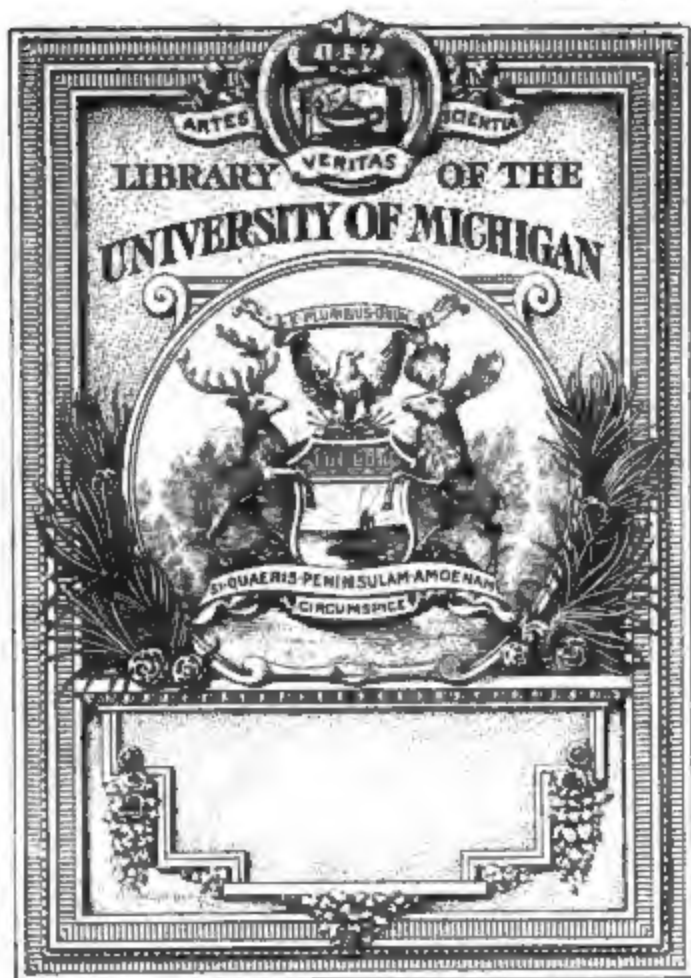


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# MEDICO-CHIRURGICAL TRANSACTIONS.

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- 1859. FREDERIC CARPENTER SKEY.
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- 1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
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- 1877. CHARLES WEST, M.D.
- 1879. JOHN ERIC ERICHSEN.
- 1881. ANDREW WHYTE BARCLAY, M.D.
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- 1884. GEORGE JOHNSON, M.D.
- 1886. GEORGE DAVID POLLOCK.
- 1888. SIR EDWARD HENRY SIEVEKING, M.D.

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(Limited to Twelve.)

### *Elected*

- 1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.
- 1887 FLOWER, WILLIAM HENRY, C.B., LL.D., F.R.S., Director of the Natural History Department, British Museum, Cromwell road.
- 1887 FOSTER, MICHAEL, LL.D., F.R.S., Professor of Physiology in the University of Cambridge.
- 1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Corresponding Member of the French Institute; The Yews, Reigate Hill, Reigate.
- 1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; The Camp, Sunningdale.
- 1868 HUXLEY, THOMAS HENRY, LL.D., D.C.L., F.R.S., late Professor of Natural History in the Royal School of Mines; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.
- 1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Hayes, Kent.
- 1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., late Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, East Sheen, Mortlake.

*Elected*

- 1883 PARKER, WILLIAM KITCHEN, F.R.S., Crowland, Trinity road, Upper Tooting.
- 1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., F.R.S., M.P., Lucasian Professor of Mathematics in the University of Cambridge; President of the Royal Society; Lensfield Cottage, Cambridge.
- 1867 TURNER, SIR WILLIAM, LL.D., F.R.S., Professor of Anatomy in the University of Edinburgh.
- 1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Honorary Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Hind Head House, Shotter Mill, near Petersfield.

## FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

*Elected*

- 1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.
- 1883 BIGELOW, HENRY J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.
- 1887 BILLINGS, JOHN S., M.D., Surgeon U.S. Army ; Librarian, Surgeon-General's Office, Washington.
- 1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna ; 20, Alger Strasse, Vienna.
- 1883 CHARCOT, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris ; Member of the Academy of Medicine ; Quai Malaquais 17, Paris.
- 1864 DONDEES, FRANZ CORNELIUS, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.
- 1883 DUBOIS REYMOND, EMIL, M.D., Professor in Berlin ; N. W. Neue Wilhelmstrasse 15, Berlin.
- 1887 ESMARCH, FRIEDRICH, M.D., Professor of Surgery in the University of Kiel.
- 1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.
- 1873 VON HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics ; Berlin.
- 1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry Berlin.
- 1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.



*Elected*

- 1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France ; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c. ; Rue de Lille, 91, Paris.
- 1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France (Academy of Sciences).
- 1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.
- 1856 VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin ; Corresponding Member of the Academy of Sciences of the Institute of France ; 10, Schellingstrasse, Berlin.
- 1887 VON VOLKMANN, RICHARD, Professor in Halle.

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**SEPTEMBER, 1888.**

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (\*) are entitled by composition to receive the Transactions.

***Elected***

1846 \*ABERCROMBIE, JOHN, M.D.

1877 ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square.  
*Trans.* 1.

1885 ABRAHAM, PHINEAS S., M.A., M.D., Lecturer on Physiology and Histology at the Westminster Hospital; 11, Nottingham place.

1851 \*ACLAND, SIR HENRY WENTWORTH, K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad street, Oxford.

*Elected*

- 1885 ACLAND, THEODORE DYKE, M.D., Assistant Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton ; 7, Brook street, Hanover square.
- 1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic ; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street ; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.
- 1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.
- 1837 \*AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary ; Cliff Point, Lower Broughton, Manchester.
- 1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.
- 1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., F.R.S., Consulting Physician to the Leeds General Infirmary ; 6, Park square, Leeds. *Trans.* 3.
- 1879 ALLCHIN, WILLIAM HENRY, M.B., F.R.S. Ed., Physician to, and Lecturer on Medicine at, the Westminster Hospital ; 5, Chandos street, Cavendish square.
- 1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's Park ; 48, Harley street, Cavendish square. *Trans.* 2.
- 1884 ANDERSON, ALEXANDER RICHARD, Resident Surgeon, General Hospital, Nottingham.
- 1881 ANDERSON, JAMES, A.M., M.D., Assistant Physician to the London Hospital ; 84, Wimpole street, Cavendish square.
- 1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich ; 105, Gloucester place, Portman square.
- 1862 ANDREW, JAMES, M.D., *Vice-President*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital ; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. V.P. 1888. *Trans.* 1.

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- 1880 \*APPLETON, HENRY, M.D., Staines.
- 1888 ARKLE, CHARLES JOSEPH, M.B.; University College Hospital.
- 1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.
- 1851 \*BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.
- 1873 \*BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.
- 1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1886-8. *Lib. Com.* 1876-7. *Trans.* 7.
- 1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.
- 1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Honorary Physician to H. M. the Queen; Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.
- 1887 BALL, JAMES BARRY, M.D., 54, Wimpole street, Cavendish square.
- 1885 BALLANCE, CHARLES ALFRED, M.S., 56, Harley street, Cavendish square. *Trans.* 1.
- 1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.
- 1866 \*BANKS, JOHN THOMAS, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.
- 1886 BANKS, WILLIAM MITCHELL, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.

*Elected*

- 1879 BARKER, ARTHUR EDWARD JAMES, Surgeon to University College Hospital, and Assistant Professor of Clinical Surgery and Teacher of Practical Surgery at University College, London; 87, Harley street, Cavendish square. *Trans.* 5.
- 1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].
- 1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.
- 1876 BARLOW, THOMAS, M.D., B.S., Physician to University College Hospital; Physician to the Hospital for Sick Children, Great Ormond street, and Assistant Physician to the London Fever Hospital; 10, Wimpole street, Cavendish square. *Trans.* 2.
- 1881 \*BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.
- 1861 BARNES, ROBERT, M.D., 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.
- 1864 BARRATT, JOSEPH GILLMAN, M.D.
- 1880 BARROW, A. BOYCE, Assistant Surgeon to King's College Hospital, to the Westminster Hospital, and to the West London Hospital; 17, Welbeck street, Cavendish square.
- 1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.
- 1859 BARWELL, RICHARD, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 11.
- 1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 20, Queen Anne street, Cavendish square. *Referee*, 1886-8. C. 1885. *Trans.* 1.

*Elected*

- 1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.
- 1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.
- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.
- 1860 \*BEALEY, ADAM, M.D., M.A., Oak Lea, Harrogate.
- 1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.
- 1871 BECK, MARCUS, M.S., Professor of Surgery in University College, London, and Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. C. 1886-7. *Referee*, 1882-5. *Lib. Com.* 1881-5.
- 1880 BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square. *Trans.* 1.
- 1871 BELLAMY, EDWARD, Senior Surgeon to, and Lecturer on Surgery at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; Examiner in Surgery in the Victoria University, Manchester; 17, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1879-81. *Trans.* 1.
- 1847 BENNET, JAMES HENRY, M.D., Mentone, Alpes Maritimes, France.
- 1880 BENNETT, ALEX. HUGHES, M.D., Physician to the Westminster Hospital; 76, Wimpole street, Cavendish square. *Trans.* 1.
- 1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to the Dental Hospital of London; 17, George street, Hanover square.



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- 1877 BENNETT, WILLIAM HENRY, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 1, Chesterfield street, Mayfair. *Trans.* 2.
- 1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.
- 1885 BERRY, JAMES, Assistant Demonstrator of Anatomy, St. Bartholomew's Hospital; 60, Welbeck street, Cavendish square.
- 1820 BERTIN, STEPHEN, Paris.
- 1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.
- 1865 \*BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.
- 1878 BINDON, WILLIAM JOHN VEREKER, M.D., 48, St. Ann's street, Manchester.
- 1856 †BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.
- 1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.
- 1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-3. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.
- 1866 BISHOP, EDWARD, M.D.
- 1881 BISS, CECIL YATES, M.D., Assistant Physician to the Middlesex Hospital, and to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.

*Elected*

- 1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada ; 6, Palace street, Quebec, Canada east.
- 1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital ; 71, Grosvenor street. C. 1883-4.
- 1846 †BOSTOCK, JOHN ASHTON, C.B., *Treasurer* ; Hon. Surgeon to H.M. the Queen ; Deputy Surgeon-General ; 73, Onslow gardens, Brompton. C. 1861-2. V.P. 1870-71. T. 1888. *Sci. Com.* 1867.
- 1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City] ; Arcachon, France.
- 1882 BOWLBY, ANTHONY ALFRED, Surgical Registrar to St. Bartholomew's Hospital ; 75, Warrington crescent, Maida hill. *Trans.* 2.
- 1870 \*BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.
- 1841 †BOWMAN, SIR WILLIAM, BART., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields ; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.
- 1886 BOXALL, ROBERT, M.D., Physician to the General Lying-in Hospital ; 6, Nottingham terrace, York Gate, Regent's Park.
- 1884 BOYD, STANLEY, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, Charing Cross Hospital ; 27, Gower street.
- 1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.
- 1874 BRADSHAW, A. F., Surgeon-Major ; Surgeon to the Rt. Hon. the Commander in Chief in India ; Army Head Quarters, Bengal Presidency. [Agent : Vesey W. Holt, 17, Whitehall place.]
- 1883 BRADSHAW, JAMES DIXON, M.B., 30, George Street, Hanover square.

*Elected*

- 1867 \*BRETT, ALFRED T., M.D., Watford, Herts.
- 1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.
- 1867 BRIDGEWATER, THOMAS, M.B., Harrow-on-the-Hill, Middlesex.
- 1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 34, Seymour street, Portman square. C. 1885. *Referee*, 1881-4. *Trans.* 5.
- 1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.
- 1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.
- 1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.
- 1888 BROWNE, HENRY LANGLEY, Moor House, West Bromwich.
- 1878 BROWNE, SIR JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; 7, Cumberland terrace, Regent's Park.
- 1880 BROWNE, JAMES WILLIAM, M.B., 7, Norland place, Holland Park.
- 1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.
- 1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Casualty Physician to St. Bartholomew's Hospital and Physician to the Royal Hospital for Diseases of the Chest; 6A, Bedford square.

*Elected*

- 1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Referee*, 1886-8. *Lib. Com.* 1888. *Trans.* 1.
- 1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888. *Referee*, 1880-87. *Lib. Com.* 1882-7.
- 1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Corresponding Member "Société de Chirurgie, Paris;" 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 10. *Pro.* 1.
- 1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingham place, Marylebone road.
- 1864 BUCKLE, FLEETWOOD, M.D.
- 1881 BULLER, AUDLEY CECIL, M.D., Oxford and Cambridge Club, Pall Mall.
- 1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 35, Brook street, Grosvenor square.
- 1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Trans.* 3.
- 1871 BUTT, WILLIAM F., 48, Park street, Park lane.
- 1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the Hospital for Women, Soho Square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.

*Elected*

- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*. 1887-8.
- 1851 \*CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.
- 1885 CAHILL, JOHN, 12, Seville street, Lowndes square.
- 1887 CALVERT, JAMES, M.D., St. Bartholomew's Hospital.
- 1888 CARLESS, ALBERT, M.B., B.S., 50, Torrington square.
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.
- 1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.
- 1888 CARTER, WILLIAM JEFFREYS BECHER, Cancer Hospital, Brompton.
- 1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital. C. 1860-1. *Sci. Com.* 1863.
- 1879 CARTWRIGHT, S. HAMILTON, late Professor of Dental Surgery at King's College, London, and Surgeon Dentist to King's College Hospital.
- 1888 CAUTLEY, EDMUND, M.B., B.C., St. Bartholomew's Hospital.
- 1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Lib. Com.* 1888. *Trans.* 1.
- 1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.

*Elected*

- 1884 CHAFFEY, WAYLAND CHARLES, M.B.; 8, North street, Brighton.
- 1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York Gate, Regent's Park. C. 1872-3.
- 1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital and to the Lock Hospital; Shrubs Hill House, Sunningdale. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.
- 1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 60, Great Cumberland place. *Lib. Com.* 1885-8. *Trans.* 7.
- 1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.
- 1885 CHAPMAN, PAUL MORGAN, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.
- 1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 9, Albert Mansions, Victoria street, Westminster.
- 1881 \*CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple row, Birmingham. *Trans.* 2.
- 1868 CHEADLE, WALTER BUTLER, M.D., *Secretary, Trustee*; Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Senior Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. *Referee*, 1885. *Trans.* 1.
- 1879 CHEYNE, WILLIAM WATSON, M.B., Assistant Surgeon to King's College Hospital, and Demonstrator of Surgery in King's College, London; 59, Welbeck street, Cavendish square. *Lib. Com.* 1886-8.
- 1873 \*CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.



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- 1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.
- 1872 CHRISTIE, THOMAS BEITH, M.D., C.I.E., Medical Superintendent, Royal India Asylum, Ealing.
- 1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. *Referee*, 1874-81.
- 1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., F.R.S., *Trustee*, *Vice-President*, Physician to, and Emeritus Professor of Clinical Medicine at, the London Hospital; 16, Cavendish square. C. 1875. V.P. 1888.
- 1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.
- 1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.
- 1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Miller Hospital, and Senior Assistant Surgeon to the Central London Ophthalmic Hospital; 21, Lee terrace, Blackheath.
- 1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.
- 1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.
- 1842 †CLAYTON, SIR OSCAR MOORE PASSEY, C.B., Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.
- 1879 †CLUTTON, HENRY HUGH, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

*Elected*

- 1857 COATES, CHARLES, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.
- 1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 8, Suffolk street, Pall Mall. *Trans.* 2.
- 1885 COLLINS, WILLIAM MAUNSELL, M.D., 10, Cadogan place.
- 1865 COOPER, ALFRED, Consulting Surgeon to the West London Hospital; Surgeon to the Lock Hospital and to St. Mark's Hospital; 9, Henrietta street, Cavendish square.
- 1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.
- 1860 \*CORRY, THOMAS CHARLES STEUART, M.D., Ormean Terrace, Belfast.
- 1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.
- 1860 †COUPER, JOHN, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882-3.
- 1877 COUPLAND, SIDNEY, M.D., Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; 14, Weymouth street, Portland place.
- 1862 †COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.
- 1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.
- 1868 CRAWFORD, SIR THOMAS, K.C.B., M.D., Hon. Surgeon to H.M. the Queen; Director General, Army Medical Department; 2, Victoria street, Westminster, and 5, St. John's Park, Blackheath. C. 1887.
- 1869 \*CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.
- 1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. *Trans.* 1.

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- 1892 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 2.
- 1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. C. 1884. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 1.
- 1862 CROMPTON, SAMUEL, M.D., Brookmead, Cranleigh, Surrey.
- 1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.
- 1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.
- 1888 CULLINGWORTH, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 46, Brook street, Grosvenor square.
- 1879 CUMBERBATCH, A. ELKIN, Aural Surgeon to St. Bartholomew's Hospital; Aural Surgeon to the Great Northern Hospital; 17, Queen Anne street.
- 1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square. *Referee*, 1884-8.
- 1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.
- 1886 DAKIN, WILLIAM RADFORD, M.D., 57, Welbeck street, Cavendish square.
- 1872 DALBY, SIR WILLIAM BARTLETT, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 18, Savile row. *Trans.* 3.
- 1884 DALLAWAY, DENNIS, 5, Duchess street, Portland place.
- 1877 DARBISHIRE, SAMUEL DUKINFELD, M.D., Physician to the Radcliffe Infirmary, Oxford; Beaumont street, Oxford.
- 1879 DARWIN, FRANCIS, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.
- 1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.
- 1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

*Elected*

- 1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.
- 1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.
- 1878 DAVY, RICHARD, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square. *Trans.* 1.
- 1882 \*DAWSON, YELVERTON, M.D., Heathlands, Southbourne-on-Sea, Hants.
- 1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.
- 1878 DENT, CLINTON THOMAS, Assistant Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. *Trans.* 2.
- 1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. C. 1874-5. V.P. 1887. *Referee*, 1869-73, 1882-6. *Sci. Com.* 1867-79. *Trans.* 13.
- 1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.
- 1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Strete place, Bournemouth. *Trans.* 2.
- 1845 DODD, JOHN.
- 1888 DONELAN, JAMES, M.B., M.C., 4, Argyll street, Regent street.
- 1879 DONKIN, HORATIO, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.

*Elected*

- 1877 DORAN, ALBAN HENRY GRIFFITHS, Assistant Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. *Trans.* 1.
- 1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.
- 1867 DRAGE, CHARLES, M.D., Hatfield, Herts.
- 1884 DRAGE, LOVELL, M.B., B.S., The Small House, Hatfield, Herts.
- 1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.
- 1885 DRUMMOND, DAVID, M.D., 7, Saville Place, Newcastle-on-Tyne.
- 1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.
- 1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 23, Sackville street, Piccadilly.
- 1865 †DUCKWORTH, SIR DYCE, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee* 1885-8. *Trans.* 1.
- 1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.
- 1845 DUFF, GEORGE, M.D., High street, Elgin.
- 1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.
- 1871 DUKE, BENJAMIN, Windmill House, Clapham Common.
- 1871 \*DUKES, CLEMENT, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.
- 1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

*Elected*

- 1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.
- 1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; 71, Brook street, Grosvenor square. *Referee*, 1881-8. *Trans.* 1.
- 1884 DUNCAN, WILLIAM, M.D., Assistant Obstetric Physician and Teacher of Operative Midwifery, Middlesex Hospital; 6, Harley street, Cavendish square.
- 1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon and Pathologist at the West London Hospital; 11, Nottingham place.
- 1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. V. P. 1887. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.
- 1874 DURHAM, FREDERIC, M.B., 82, Brook street, Grosvenor square.
- 1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.
- 1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.
- 1887 EASMON, JOHN FARRELL, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.
- 1868 EASTES, GEORGE, M.B.Lond., 69, Connaught street, Hyde Park square.
- 1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 2.
- 1883 EDWARDES, EDWARD JOSHUA, M.D., 16, Acacia road, St. John's Wood.
- 1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 93, Wimpole street, Cavendish square.

*Elected*

- 1824 EDWARDS, GEORGE.
- 1869 ELAM, CHARLES, M.D., 75, Harley street, Cavendish square.  
*Lib. Com.* 1886-8.
- 1887 ELLIOTT, JOHN, 16, Heathcote street, Gray's Inn Road.
- 1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University College, London; Minsterworth, Gloucester.  
C. 1863-4. *Trans.* 2.
- 1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.
- 1854 \*ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.
- 1842 †ERICHSEN, JOHN ERIC, LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C. 1855-6. V.P. 1868. P. 1879-80.  
*Referee*, 1866-8, 1884-7. *Lib. Com.* 1844-7, 1854.  
*Trans.* 2.
- 1879 EVE, FREDERIC S., Pathological Curator of the Museum, Royal College of Surgeons; Assistant Surgeon to the London Hospital; 15, Finsbury circus. *Trans.* 2.
- 1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. *Trans.* 1.
- 1875 \*FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.
- 1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.
- 1862 FARQUHARSON, ROBERT, M.D., LL.D., M.P., Migvie Lodge, Porchester gardens, Hyde park; Finzean, Aboyne, Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

*Elected*

- 1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; late Surgeon-General Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. C. 1888. *Referee*, 1881-7.
- 1887 FEENY, MICHAEL HENRY, Les Avants, Montreux, Switzerland.
- 1872 \*FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.
- 1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-8. *Trans.* 4.
- 1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Forensic Medicine in King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Trans.* 2.
- 1852 \*FIELD, ALFRED GEORGE.
- 1849 †FINCHAM, GEORGE TUPMAN, M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.
- 1879 FINLAY, DAVID WHITE, M.D., Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 9, Lower Berkeley street, Portman square. *Trans.* 2.
- 1866 FISH, JOHN CROCKETT, B.A., M.D., 92, Wimpole street, Cavendish square.
- 1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park.
- 1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.



*Elected*

- 1864 \*FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.
- 1877 DE FONMARTIN, HENRY, M.D., Parkhurst, Isle of Wight.
- 1848 †FORBES, JOHN GREGORY, Ellerslie, Widmore road, Bromley, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.
- 1865 FOSTER, SIR BALTHAZAR WALTER, M.D., M.P., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 14, Temple row, Birmingham.
- 1883 FOWLER, JAMES KINGSTON, M.A., M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.
- 1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.
- 1887 FOX, RICHARD HINGSTON, M.D., 43, Finsbury circus.
- 1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.
- 1871 FRANK, PHILIP, M.D., Cannes, France.
- 1884 \*FRANKS, KENDAL, M.D., Surgeon to the Adelaide Hospital and to the Throat and Ear Hospital, Dublin; 69, Fitzwilliam square, Dublin.
- 1843 FRASER, PATRICK, M.D., C. 1866.
- 1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.
- 1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.
- 1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, May Fair.
- 1876 FURNER, WILLOUGHBY, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

*Elected*

- 1864 \*GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow.
- 1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. *Referee*, 1882-8. *Lib. Com.* 1883-4. *Trans.* 2.
- 1883 GALTON, JOHN CHARLES, M.A., F.L.S., 45, Great Marlborough street.
- 1885 GAMGEE, ARTHUR, M.D., F.R.S., Assistant Physician to St. George's Hospital; 17, Great Cumberland place, Hyde Park.
- 1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. *Referee*, 1886-8. *Lib. Com.* 1882-5. *Trans.* 3.
- 1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.
- 1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.
- 1854 †GARROD, SIR ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.
- 1886 GARROD, ARCHIBALD EDWARD, M.A., M.D., Casualty Physician to St. Bartholomew's Hospital, and Physician to the Marylebone Dispensary; 9, Chandos street, Cavendish square. *Trans.* 2.
- 1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.
- 1819 GAULTER, HENRY.
- 1887 GAY, JOHN, 119, Upper Richmond road, Putney.
- 1866 GEE, SAMUEL JONES, M.D., *Librarian*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Consulting Physician to the Hospital for Sick Children; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887-8. *Sci. Com.* 1879. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.

*Elected*

- 1885 GELL, HENRY WILLINGHAM, M.B., 43, Albion street, Hyde Park.
- 1878 GERVIS, HENRY, M.D., Consulting Obstetric Physician to St. Thomas's Hospital; Consulting Physician to the Royal Maternity Charity; 40, Harley street, Cavendish square. *Referee*, 1884-8. *Trans.* 1.
- 1884 GIBBES, HENEAGE, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.
- 1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place.
- 1877 GODLEE, RICKMAN JOHN, Surgeon to University College Hospital, and Teacher of Operative Surgery in University College, London; Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Consumption, Brompton; 81, Wimpole street, Cavendish square. *Referee*, 1886-8. *Trans.* 3.
- 1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.
- 1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Assistant Surgeon and Lecturer on Physiology at Guy's Hospital; 13, St. Thomas street, Southwark.
- 1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.
- 1883 GOODHART, JAMES FREDERIC, M.D., Physician to, and Lecturer on Pathology at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.
- 1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; 16, Queen Anne street, Cavendish square. *Trans.* 2.

*Elected*

- 1873 GOWERS, WILLIAM RICHARD, M.D., F.R.S., Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Referee* 1888. *Lib. Com.* 1884-6. *Trans.* 7.
- 1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.
- 1868 GREEN, T. HENRY, M.D., Physician to Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.
- 1875 \*GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.
- 1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.
- 1882 GRESSWELL, DAN ASTLEY, M.B., 5, Oakley square, Hampstead road.
- 1885 GRIFFITH, WALTER SPENCER ANDERSON, M.B., Physician to the Samaritan Free Hospital for Women and Children; 114, Harley street, Cavendish square.
- 1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; 27, Curzon street, Mayfair.
- 1852 GROVE, JOHN, Fyning, Austen road, Guildford.
- 1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.
- 1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to H.M. the Queen; and Physician in Ordinary to H.R.H. the Prince of Wales; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

*Elected*

- 1885 GULLIVER, GEORGE, M.B., Assistant Physician to, and Lecturer on Comparative Anatomy at, St. Thomas's Hospital; 16, Welbeck street.
- 1883 GUNN, ROBERT MARCUS, M.B., Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street; 54, Queen Anne street, Cavendish square.
- 1886 HABERSHON, SAMUEL HERBERT, M.D., Casualty Physician to St. Bartholomew's Hospital; 2, Upper Wimpole street, Cavendish square.
- 1854 †HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881-2. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.
- 1888 HADDEN, WALTER BAUGH, M.D., Assistant Physician and Demonstrator of Morbid Anatomy at St. Thomas's Hospital; Assistant Physician, Hospital for Sick Children; 21, Welbeck street, Cavendish square.
- 1885 HAIG, ALEXANDER, M.D., Casualty Physician to St. Bartholomew's Hospital; 30, Welbeck street, Cavendish square. *Trans.* 3.
- 1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square.
- 1885 HALLIBURTON, WILLIAM DOBINSON, M.D., Assistant Professor of Physiology, University College, London; 25, Maitland Park Villas, Haverstock Hill.
- 1870 HAMILTON, ROBERT, Surgeon to the Royal Southern Hospital, Liverpool; 1 Prince's road, Liverpool.
- 1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.
- 1856 †HARE, CHARLES JOHN, M.D., *Treasurer*, late Professor of Clinical Medicine in University College, London, and Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887.

*Elected*

- 1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.
- 1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-8. *Sci. Com.* 1879. *Trans.* 10.
- 1880 HARRIS, VINCENT DORMER, M.D., Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 31, Wimpole street, Cavendish square.
- 1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Victoria University; 41, Rodney street, Liverpool. *Trans.* 1.
- 1854 HAVILAND, ALFRED.
- 1870 HAWARD, J. WARRINGTON, *Secretary*; Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888. *Lib. Com.* 1881-4. *Trans.* 2.
- 1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 9, Duke street, Portland place. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.
- 1885 HAWKINS, FRANCIS HENRY, M.B., Physician to St. George's and St. James's Dispensary and to the North London Hospital for Consumption; 22, Henrietta street, Cavendish square.
- 1848 †HAWKSLEY, THOMAS, M.D., 11, Albert Mansions, Victoria street, and Beomands, Chertsey, Surrey.
- 1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.
- 1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

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- 1861 HAYWARD, WILLIAM HENRY.
- 1848 \*HEALE, JAMES NEWTON, M.D.
- 1865 HEATH, CHRISTOPHER, *Trustee*, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 3.
- 1850 HEATON, GEORGE, M.D., Boston, U.S.
- 1882 HENSLEY, PHILIP JOHN., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.
- 1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Trans.* 1.
- 1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.
- 1883 HERRINGHAM, WILMOT PARKER, M.D., 22, Bedford square.
- 1843 †HEWETT, SIR PRESCOTT GARDNER, Bart., F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Foreign Associate of the "Académie de Médecine," and Corresponding Member of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.
- 1887 HEWITT, FREDERIC WILLIAM, M.D., 10, George street, Hanover square.
- 1855 †HEWITT, W. M. GRAILY, M.D., Emeritus Professor of Midwifery in University College, London, and Consulting Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-88. *Lib. Com.* 1868, 1874.
- 1880 HICKS, CHARLES CYRIL, M.D., Wokingham, Berks.
- 1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

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- 1862 HILL, M. BERKELEY, M.B., Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. *Referee*, 1888. C. 1878-9. S. 1881-4. V.P. 1885-6. *Trans.* 1.
- 1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.
- 1861 \*HOFFMEISTER, SIR WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.
- 1843 †HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.
- 1879 HOLLAND, PHILIP ALEXANDER, M.A.
- 1868 HOLLIS, WILLIAM AINSLIE, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.
- 1861 HOLMAN, WILLIAM HENRY, M.B., 68, Adelaide road, South Hampstead.
- 1856 †HOLMES, TIMOTHY, M.A., Consulting Surgeon to St. George's Hospital; Corresponding Member of the "Société de Chirurgie," Paris; 18, Great Cumberland place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.
- 1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster, 14, Savile row, Burlington Gardens. C. 1862-3. V.P. 1879-80.
- 1846 †HOLTHOUSE, CARSTEN, 5, Bloomsbury square. C. 1863. *Referee* 1870-6. *Lib. Com.* 1859-60.
- 1878 HOOD, DONALD WILLIAM CHARLES, M.D., Senior Physician to the North-West London Hospital; Physician to the West London Hospital; 43, Green street, Park lane.



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- 1883 HORSLEY, VICTOR ALEXANDER HADEN, F.R.S., Assistant Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; Professor of Pathology in University College, London; Superintendent of the Brown Institution, Wandsworth road; 80, Park street, Grosvenor Square. *Trans.* 1.
- 1865 HOWARD, BENJAMIN, M.D. [New York, U.S.] *Trans.* 1.
- 1881 HOWARD, HENRY, M.B., abroad. [6, The Terrace, Mount Pleasant, Cambridge.]
- 1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. *Sci.Com.* 1879. *Referee*, 1887-8. *Trans.* 2.
- 1886 HUDSON, CHARLES LEOPOLD, Pathologist and Curator of the Museum, Middlesex Hospital; Warden, Medical College, Cleveland street.
- 1884 HUGGARD, WILLIAM R., M.D. [Place de la Synagogue, 2, Genève.]
- 1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-88. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 9.
- 1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.
- 1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 6.
- 1882 HUMPHRY, LAURENCE, M.B., 3, Trinity street, Cambridge.
- 1873 HUNTER, SIR W. GUYER, M.D., M.P., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.
- 1849 HUSSEY, EDWARD LAW, Consulting Surgeon to the Oxford County Lunatic Asylum and the Warneford Asylum; 24, Winchester Road, Oxford. *Trans.* 1.

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- 1856 †HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883-8. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.
- 1888 HUTCHINSON, JONATHAN, Jun., 15, Cavendish square.
- 1820 HUTCHINSON, WILLIAM, M.D.
- 1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.
- 1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]
- 1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.
- 1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.
- 1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road, south, Wolverhampton.
- 1883 JACOBSON, WALTER HAMILTON ACLAND, B.A., M.B., M.S., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 41, Finsbury square. *Trans.* 1.
- 1825 JAMES, JOHN B., M.D.
- 1883 \*JENKINS, EDWARD JOHNSTONE, M.D., The Australian Club, Sydney, New South Wales.
- 1851 †JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

*Elected*

- 1884 JENNINGS, CHARLES EGERTON, M.S., M.B., 15, Upper Brook street, Grosvenor square.
- 1881 JENNINGS, WILLIAM OSCAR, M.D., 35, Rue Marboëuf, Avenue des Champs-Élysées, Paris.
- 1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.
- 1883 JESSOP, WALTER H. H., M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.
- 1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Gènevois."
- 1847 †JOHNSON, GEORGE, M.D., F.R.S., Consulting Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884-5. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib.Com.* 1860-1. *Trans.* 10. *Pro.* 1.
- 1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Cortina, Netherhall terrace, South Hampstead, and 14, Stratford place, Oxford street.
- 1884 JOHNSTON, JAMES, M.D., 11, Chester place, Hyde Park square.
- 1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.
- 1887 JONES, HENRY LEWIS, M.D., Casualty Physician to St. Bartholomew's Hospital, 6, West street, Finsbury Circus.
- 1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.
- 1875 \*JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

*Elected*

- 1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.
- 1865 JORDAN, FURNEAUX, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.
- 1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.
- 1816 \*KAUFFMANN, GEORGE HERMANN, M.D., Hanover.
- 1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; Surgeon to the Surgical Aid Society; 10, George street, Hanover square.
- 1872 KELLY, CHARLES, M.D., Professor of Hygiene in King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.
- 1848 \*KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.
- 1884 KESER, JEAN SAMUEL, M.D., Surgeon to the French Hospital, Leicester place; 11, Harley street, Cavendish square.
- 1877 \*KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.
- 1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.
- 1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 4.
- 1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and Consulting Surgeon to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.
- 1885 KLEIN, EDWARD EMANUEL, M.D., F.R.S., Lecturer on Physiology, St. Bartholomew's Hospital; 19, Earl's Court square.

*Elected*

- 1883 KNAPTON, GEORGE, 11, Hoghton street, Southport.
- 1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon. [57, West Cromwell road.]
- 1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital. C. 1849-50. V.P. 1865. *Referee*, 1850. [Care of Ernest Lane, 10, Cambridge street, Hyde park.]
- 1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to Guy's Hospital and to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 2.
- 1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 26, Upper Wimpole street, Cavendish square.
- 1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. *Referee*, 1885-8. *Lib. Com.* 1879-80, 1888. *Trans.* 1.
- 1873 \*LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- 1816 LAWRENCE, G. E.
- 1888 LAWRENCE, LAURIE ASHER, 134, Harley street.
- 1884 LAWSON, GEORGE, Surgeon-Oculist to H.M. the Queen; Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street, Cavendish square.
- 1880 LAYCOCK, GEORGE LOCKWOOD, M.B., Physician to the Paddington Green Children's Hospital; 42, Manchester street, Manchester square.

*Elected*

- 1886 \*LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary ; 41, Lowther street, Carlisle.
- 1882 LEDWICH, EDWARD L'ESTRANGE, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin ; 23, Upper Leeson Street, Dublin.
- 1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital ; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.
- 1884 LEE, ROBERT JAMES, M.D., 6, Savile row.
- 1883 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden road, Twickenham.
- 1869 LEGG, JOHN WICKHAM, M.D., Hotel Beauséjour, Cannes. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.
- 1836 LEIGHTON, FREDERICK, M.D.
- 1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., 60, Wimpole street, Cavendish square.
- 1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital ; Paris.
- 1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen ; Professor of Clinical Surgery at King's College, London ; and Surgeon to King's College Hospital ; 12, Park crescent, Regent's Park.
- 1872 \*LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester ; 21, St. John street, Manchester.
- 1871 LITTLE, LOUIS STROMEYER, Shanghai, China.
- 1819 LLOYD, ROBERT, M.D.
- 1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.
- 1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital, and Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital ; 19, Upper Berkeley street. *Trans.* 1. .

*Elected*

- 1860 LONGMORE, SIR THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Foreign Associate "Académie de Médecine;" Woolston Lawn, Woolston, Hants. *Trans.* 2.
- 1836 LÖWENFELD, JOSEPH S., M.D., Berbice.
- 1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.
- 1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.
- 1888 LUFF, ARTHUR PEARSON, M.B., B.Sc., 35, Westbourne terrace, Hyde Park.
- 1883 LUND, EDWARD, Professor of Surgery, and Member of Senate, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.
- 1887 LUSH, PERCY J. F., M.B., Fitzjohn's Mansions, South Hampstead.
- 1857 LYON, FELIX WILLIAM, M.D., 7, South Charlotte street, Edinburgh.
- 1867 MABERLY, GEORGE FREDERICK, Mailai Valley, Nelson, New Zealand.
- 1873 MACCARTHY, JEREMIAH, M.A., Surgeon to the London Hospital and Lecturer on Physiology at the London Hospital Medical College; 15, Finsbury square. C. 1886-7. *Lib. Com.* 1882-5.
- 1867 MAC CORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. C. 1884-5. *Trans.* 1.
- 1887 MACDONALD, GEORGE CHILDS, M.D.

*Elected*

- 1862 \*M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Jervis street Hospitals ; 89, Merrion square west, Dublin. *Trans.* 2.
- 1880 MACFARLANE, ALEXANDER WILLIAM, M.D., Examiner in Medical Jurisprudence, University of Glasgow ; 6, Manchester Square.
- 1866 MACGOWAN, ALEXANDER THORBURN, M.D., Vyvyan House, Clifton, near Bristol.
- 1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London ; Surgeon to the Royal South London Ophthalmic Hospital ; 5, Savile row.
- 1822 MACINTOSH, RICHARD, M.D.
- 1859 \*M'INTYRE, JOHN, M.D., Odiham, Hants.
- 1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Surgeon to St. Thomas's Hospital ; Surgeon-in-Chief to the Metropolitan Police Force ; [2, Chandos street, Cavendish square].
- 1881 MACKENZIE, STEPHEN, M.D., Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College ; Physician to the Royal London Ophthalmic Hospital ; 26, Finsbury square. *Trans.* 1.
- 1885 MACKERN, JOHN, M.D., Assistant Physician to the Chelsea Hospital for Women ; 30, Cambridge street, Hyde park.
- 1876 MACKEY, EDWARD, M.D., Assistant Physician to the Sussex County Hospital ; 1, Brunswick road, Hove, Brighton.
- 1854 \*MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.
- 1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein ; 9, Cadogan place, Belgrave square.



*Elected*

- 1876 MACNAMARA, CHARLES N., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. *Referee*, 1884-8. *Lib. Com.* 1886-8.
- 1881 MACREADY, JONATHAN FORSTER CHRISTIAN HORACE, Surgeon to the Great Northern Hospital; 51, Queen Anne street, Cavendish square.
- 1880 MADDICK, EDMUND DISTIN, 2, Chandos street, Cavendish square.
- 1886 MAGUIRE, ROBERT, M.D., 4, Seymour street, Portman square.
- 1880 MAKINS, GEORGE HENRY, Assistant Surgeon to St. Thomas's Hospital and to the Evelina Hospital for Children; 2, Queen street, May Fair. *Trans.* 1.
- 1885 MALCOLM, JOHN DAVID, M.B., Surgeon in charge of Out-Patients, Samaritan Free Hospital; 24, Bryanston street, Portman square. *Trans.* 1.
- 1876 MALLAM, BENJAMIN, Rose Bank, Blackall road, Exeter.
- 1855 MARCET, WILLIAM, M.D., F.R.S., Flowermead, Wimbledon Park, and Athenæum Club, Pall Mall. C. 1871. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.
- 1867 MARSH, F. HOWARD, Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children, Great Ormond street; 30, Bruton street, Berkeley square. C. 1882-3. S. 1885-7. *Lib. Com.* 1880-1. *Trans.* 4.
- 1851 †MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1882-3. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.
- 1884 MARTIN, SIDNEY HARRIS COX, M.D.; 60, Gower street.

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- 1883 MAUDSLEY, HENRY CARR, M.D., 11, Spring street, Melbourne, Victoria.
- 1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary ; Bradford, Yorkshire. *Trans.* 1.
- 1865 MEDWIN, AARON GEORGE, M.D., Consulting Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square, and 11, Montpelier row, Blackheath.
- 1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children ; 6, Queen Anne street, Cavendish square.
- 1874 MERRIMAN, JOHN J., 45, Kensington square.
- 1815 MEYER, AUGUSTUS, M.D., St. Petersburg.
- 1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital ; The Limes, Bristol road, Edgbaston, Birmingham.
- 1854 MIDDLESHP, EDWARD ARCHIBALD.
- 1885 MILLICAN, KENNETH WILLIAM, B.A., 58, Welbeck street.
- 1882 MILLS, JOSEPH, 15, Henrietta street, Cavendish square.
- 1873 MILNER, EDWARD, Surgeon to the Lock Hospital ; 32, New Cavendish street, Portland place.
- 1887 MIVART, FREDERICK ST. GEORGE, Beaumont, Worple road, Wimbledon.
- 1883 MONEY, ANGEL, M.D., Assistant Physician to University College Hospital, to the Hospital for Sick Children, Great Ormond Street, and to the City of London Hospital for Diseases of the Chest, Victoria Park ; 24 Harley street, Cavendish square. *Trans.* 4.
- 1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College, and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital ; The Warden's House, St. Bartholomew's Hospital. *Referee*, 1886-8.
- 1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

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- 1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Victoria University, Manchester ; 1, St. Peter's square, Manchester.
- 1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital and to the Hospital for Sick Children, Great Ormond street ; 68, Grosvenor street. *Trans.* 1.
- 1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital ; 2, Mansfield street, Portland place. C. 1888. *Referee*, 1882-7. *Trans.* 10.
- 1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital ; 8, Harley street, Cavendish square.
- 1885 MOTT, FREDERICK WALKER, M.D., Lecturer on Physiology, Charing Cross Hospital ; Meadowlead, Gayton road, Harrow.
- 1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians ; Consulting Physician to the Royal Hospital for Incurables ; 40, Finsbury square.
- 1873 MURRAY, J. IVOR, M.D., F.R.S.Ed. 24, Huntriss row, Scarborough.
- 1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest ; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital ; 38, Weymouth street, Portland place. *Trans.* 1.
- 1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards ; 3, Park Terrace, Windsor. C. 1878-9. *Lib. Com.* 1877.
- 1882 MYERS, ARTHUR THOMAS, M.D., Medical Registrar, St. George's Hospital ; 9, Lower Berkeley street, Portman square.
- 1881 NALL, SAMUEL, M.B., Disley, Stockport, Cheshire.
- 1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne ; 166, Collins street east, Melbourne, Victoria.

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- 1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York Gate, Regent's Park. *Lib. Com.* 1841.
- 1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square.
- 1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.
- 1868 NICHOLLS, JAMES, M.D., Senior Medical Officer, Essex and Chelmsford Infirmary and Dispensary; the Old Infirmary, Chelmsford, Essex.
- 1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.
- 1847 \*NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Exeter.
- 1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.
- 1884 OAKES, ARTHUR, M.D., 99, Priory road, West Hampstead.
- 1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption; Greenhill Park, Harlesden.
- 1847 O'CONNOR, THOMAS, March, Cambridgeshire.
- 1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 13, Welbeck street, Cavendish square.
- 1880 OGILVIE, LESLIE, M.B., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.
- 1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. V.P. 1886. *Referee*, 1864-72. *Trans.* 4.

*Elected*

- 1855 \*OGLE, WILLIAM, M.A., M.D., Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.
- 1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. V.P. 1887. *Lib. Com.* 1871-5. *Trans.* 5.
- 1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].
- 1883 \*OLIVER, THOMAS, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 12, Eldon square, Newcastle-upon-Tyne.
- 1871 \*O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.
- 1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. *Referee*, 1884-8. *Trans.* 6.
- 1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street. *Trans.* 1.
- 1885 ORMSBY, L. HEPENSTAL, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.
- 1879 OWEN, EDMUND, Joint Lecturer on Surgery at St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.
- 1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 5, Hertford street, May Fair.

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- 1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. *Referee*, 1884-8. *Lib. Com.* 1886-8. *Trans.* 3.
- 1887 PAGET, CHARLES EDWARD, Kendal, Westmoreland.
- 1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; Foreign Associate of the 'Académie de Médecine,' Paris; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.
- 1886 PAGET, STEPHEN, 57, Wimpole street, Cavendish square.
- 1858 \*PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; The Old Residence, Ripon, Yorkshire.
- 1887 PARDINGTON, GEORGE LUCAS, M.D., 47, Mount Pleasant road, Tunbridge Wells.
- 1873 PARKER, ROBERT WILLIAM, Surgeon to the East London Hospital for Children; 8, Old Cavendish street. C. 1888. *Lib. Com.* 1885-7. *Trans.* 4.
- 1885 PARKER, RUSHTON, M.B., Professor of Surgery, University College, Liverpool (Victoria University); Assistant Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.
- 1883 PASTEUR, WILLIAM, M.D., Medical Registrar to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 19, Queen street, May Fair.
- 1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.
- 1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Sci. Com.* 1879. *Lib. Com.* 1878-85.

*Elected*

- 1887 PEARCE, WALTER, M.D., 63, Montagu square.
- 1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.
- 1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.
- 1830 PELECHIN, CHARLES P., M.D., St. Petersburg.
- 1855 \*PEMBERTON, OLIVER, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.
- 1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.
- 1887 PENROSE, FRANCIS GEORGE, M.D., Colebyfield, Wimbledon.
- 1879 \*PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.
- 1878 \*PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.
- 1883 PHILLIPS, CHARLES DOUGLAS F., M.D., F.R.S.Ed., 10, Henrietta street, Cavendish square, W.
- 1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Leinster square, Bayswater.
- 1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. *Referee*, 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.
- 1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.
- 1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 10, St. Thomas's street, Southwark.
- 1885 POLAND, JOHN, Demonstrator of Anatomy, Guy's Hospital; 16, St. Thomas's street, Southwark.

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- 1884 POLLARD, BILTON, Assistant Surgeon and Surgical Registrar to University College Hospital, Surgeon to the North Eastern Hospital for Children; 24, Harley street, Cavendish square.
- 1871 POLLOCK, ARTHUR JULIUS, M.D., Senior Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.
- 1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. P. 1886-7. *Referee*, 1858, 1864-9, 1877-85. *Trans.* 5.
- 1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.
- 1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street. *Referee*, 1887-8. *Trans.* 2.
- 1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.
- 1846 POTTER, JEPHSON, M.D., F.L.S.
- 1842 POWELL, JAMES, M.D.
- 1867 POWELL, RICHARD DOUGLAS, M.D., Physician Extraordinary to H.M. the Queen, Physician to, and Lecturer on Practical Medicine at, the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole st., Cavendish square. S. (Oct.), 1883-5. C. 1887-8. *Referee* 1879-83, 1886. *Trans.* 2.
- 1887 POWER, D'ARCY, M.B., 26, Bloomsbury square.
- 1867 POWER, HENRY, Senior Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.



*Elected*

- 1857 PRIESTLEY, WILLIAM OVEREND, M.D., LL.D., Consulting Physician to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.
- 1883 PRINGLE, JOHN JAMES, M.B., C.M., Assistant Physician to, and Physician in Charge of Skin Department at, the Middlesex Hospital, and Physician to the Royal Hospital for Diseases of the Chest; 35, Bruton street, Berkeley square. *Trans.* 1.
- 1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.
- 1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital and to the Victoria Hospital for Children; 4, Sackville street, Piccadilly.
- 1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 54, Harley street, Cavendish square. *Lib. Com.* 1887-8.
- 1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.
- 1852 †RADCLIFFE, CHARLES BLAND, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-6. *Referee*, 1862-6, 1870-8.
- 1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square. *Referee*, 1885-8.
- 1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

*Elected*

- 1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.
- 1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.
- 1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.
- 1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.
- 1882 REID, JAMES, M.D., Resident Physician to H.M. the Queen, Windsor Castle.
- 1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury.
- 1855 †REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.
- 1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.
- 1881 RICE, GEORGE, M.B., C.M., Sutton, Surrey.
- 1887 RICHARDSON, GILBERT, M.D., Thornton, Upper Richmond road, Putney.
- 1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.
- 1863 RINGER, SYDNEY, M.D., F.R.S., Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 6.
- 1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury square. C. 1885-6. *Trans.* 4.
- 1871 \*ROBERTS, DAVID LLOYD, M.D., Obstetric Physician to the Manchester Royal Infirmary, Physician to St. Mary's Hospital, Manchester; 11, St. John street, Manchester.

*Elected*

- 1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London ; and Physician to University College Hospital ; Physician to the Hospital for Consumption, Brompton ; 102, Harley street, Cavendish square.
- 1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum ; Monaghan, Ireland.
- 1873 ROBERTSON, WILLIAM HENRY, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital ; Buxton, Derbyshire.
- 1888 \*ROBINSON, FREDERICK WILLIAM, M.B., C.M., Huddersfield.
- 1885 ROCKWOOD, WILLIAM GABRIEL, M.D., Colombo, Ceylon.
- 1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity ; and to the Royal Infirmary for Children and Women, Waterloo Bridge road ; 19, Ovington Gardens. C. 1879-80.
- 1857 †ROSE, HENRY COOPER, M.D., F.L.S., Consulting Surgeon to the Hampstead Dispensary ; Penrose House, Hampstead. C. 1886-7. *Trans.* 1.
- 1883 ROSE, WILLIAM, M.B., Professor of Surgery at King's College, Surgeon to King's College Hospital and to the Royal Free Hospital ; 50, Harley street, Cavendish square.
- 1882 ROUTH, AMAND JULES McCONNEL, M.D., B.S., Physician to the Samaritan Free Hospital for Women ; Assistant Obstetric Physician to the Charing Cross Hospital ; 6, Upper Montagu street, Montagu square.
- 1849 †ROUTH, CHARLES HENRY FELIX, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children ; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.
- 1863 ROWE, THOMAS SMITH, M.D., Senior Visiting Surgeon to the Royal Sea-Bathing Infirmary ; Cecil street, Margate, Kent.

*Elected*

- 1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.
- 1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.
- 1886 SAINSBURY, HARRINGTON, M.D., Assistant Physician and Pathologist to the Royal Free Hospital; 63, Welbeck street, Cavendish square. *Trans.* 1.
- 1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.
- 1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.
- 1855 †SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.
- 1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.
- 1879 SANGSTER, ALFRED, B.A., M.B., Physician to the Skin Department, and Demonstrator of Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.
- 1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Baschurch, near Shrewsbury.
- 1869 SANSOM, ARTHUR ERNEST, M.D., Senior Physician to the North-Eastern Hospital for Children; Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. C. 1887-8. *Trans.* 2.
- 1886 SAUNDBY, ROBERT, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; 83A, Edmund street, Birmingham.

*Elected*

- 1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales ; 13A, George street, Hanover square. C. 1872-3.
- 1834 SAUVAN, LUDWIG V., M.D., Warsaw.
- 1879 SAVAGE, GEORGE HENRY, M.D., Medical Superintendent and Resident Physician to the Bethlem Royal Hospital, St. George's road, Southwark.
- 1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon Extraordinary to H.M. the Queen, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital ; Surgeon to Christ's Hospital ; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 8.
- 1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London ; University College, Gower street. *Referee*, 1888.
- 1887 SCOTT, HARRY, M.D., 8, Queen Anne's Gate.
- 1861 \*SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary ; Waverley House, Huddersfield.
- 1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School ; 95, Oxford gardens, Notting hill.
- 1863 SEDGWICK, WILLIAM, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.
- 1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital ; 39, Wimpole street, Cavendish square. *Trans.* 1.
- 1875 SEMPLE, ROBERT HUNTER, M.D., Consulting Physician to the Bloomsbury Dispensary ; 8, Torrington square. *Sci. Com.* 1879.
- 1873 \*SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital ; the Barnfield, Exeter.

*Elected*

- 1882 SHARKEY, SEYMOUR JOHN, M.D., Assistant Physician, Joint Lecturer on Pathology, and Demonstrator of Morbid Anatomy, to St. Thomas's Hospital; 2, Portland place. *Trans.* 2.
- 1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.
- 1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.
- 1886 SHAW, LAURISTON ELGIE, M.D., Medical Registrar and Demonstrator of Practical Medicine, Guy's Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 15, St. Thomas's street, Southwark.
- 1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., Assistant Surgeon, Charing Cross Hospital; 20, Stratford place, Oxford street. *Trans.* 1.
- 1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.
- 1887 SIDEBOTHAM, EDWARD JOHN, M.B., Erlesdene, Bowdon, Cheshire.
- 1848 †SIEVEKING, SIR EDWARD HENRY, M.D., *President*, Physician in Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.
- 1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., Surgeon in charge of out-patients, St. Mary's Hospital; Assistant Surgeon, Royal London Ophthalmic Hospital; 101, Harley street, Cavendish square.
- 1842 †SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

*Elected*

- 1857 SIOBDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.
- 1879 SMITH, E. NOBLE, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary ; Orthopædic Surgeon to the British Home for Incurables ; 24, Queen Anne street, Cavendish square.
- 1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians ; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest ; 5, George street, Hanover square.
- 1866 SMITH, HEYWOOD, M.A. M.D., 18, Harley street, Cavendish square.
- 1886 SMITH, HOWARD LYON, 80, Tollington Park.
- 1885 SMITH, JAMES GREIG, M.B., C.M., F.R.S.Ed., Surgeon to the Bristol Royal Infirmary ; 16, Victoria square, Clifton, Bristol.
- 1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital ; Physician to the Royal Hospital for Diseases of the Chest, City road ; 68, Harley street, Cavendish square. *Trans.* 1.
- 1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital ; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.
- 1863 SMITH, THOMAS, *Vice-President*, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital ; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.
- 1874 \*SMITH, WILLIAM ROBERT, M.D., D.Sc., F.R.S.Ed., 74, Great Russell Street, Bloomsbury.
- 1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

*Elected.*

- 1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy ; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.
- 1844 SPACKMAN, FREDERICK ROBERT, M.D., Consulting Physician to St. Alban's Hospital, Harpenden, St. Alban's.
- 1887 SPENCER, WALTER GEORGE, M.B., Assistant Surgeon to the Westminster Hospital ; 94, Wimpole street, Cavendish square.
- 1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital ; 28, Welbeck street, Cavendish square.
- 1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.
- 1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.
- 1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption ; 23, Seymour street, Portman square. *Trans.* 1.
- 1882 STEAVENSON, WILLIAM EDWARD, M.D., Electrician to St. Bartholomew's Hospital ; Physician to the Alexandra Hospital for Children ; 39, Welbeck street, Cavendish square.
- 1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board, Whitehall ; Mitcham House, Mitcham, Surrey.
- 1884 STEWART, EDWARD, M.D., 16, Harley street, Cavendish square.
- 1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.
- 1879 \*STIRLING, EDWARD CHARLES, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital ; Adelaide, South Australia [care of T. Gemmell, Esq., 11, Essex street, Strand].
- 1856 †STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.



*Elected*

- 1865 STOKES, SIR WILLIAM, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.
- 1884 STONHAM, CHARLES, Assistant Surgeon to the Westminster Hospital, and Curator of Anatomical Museum, University College, London; 62, Welbeck street, Cavendish square.
- 1843 STORRS, ROBERT REEVE, Paris.
- 1871 STRONG, HENRY JOHN, M.D., Surgeon to the Croydon General Hospital; Whitgift House, George street, Croydon.
- 1863 †STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1882-8.
- 1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.
- 1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital; 9, Finsbury square. *Referee*, 1888. *Trans.* 1.
- 1883 SUTTON, JOHN BLAND, Assistant Surgeon, Lecturer on Comparative Anatomy, and Senior Demonstrator of Anatomy to the Middlesex Hospital; 22, Gordon street, Gordon square. *Trans.* 4.
- 1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to Guy's Hospital; 26, Weymouth street, Portland place.
- 1878 \*SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.
- 1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.
- 1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.
- 1875 TAY, WARREN, Surgeon to the London Hospital, to the Royal London Ophthalmic Hospital, to the North Eastern Hospital for Children, and to the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

*Elected*

- 1873 TAYLOR, FREDERICK, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Referee*, 1887-8. *Trans.* 1.
- 1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's Wood.
- 1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.
- 1859 TEGART, EDWARD, 49, Jermyn street, St. James's. C. 1888.
- 1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.
- 1862 THOMPSON, EDMUND SYMES, M.D., Senior Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Referee*, 1876-7. *Trans.* 1.
- 1852 †THOMPSON, SIR HENRY, *Vice-President*, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the "Société de Chirurgie," Paris; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 7.
- 1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.
- 1881 THOMSON, WILLIAM SINCLAIR, M.D., late Senior Consulting Surgeon to Peterbro' Hospital, and Medical Officer of Health for Peterbro'; 40, Ladbroke grove, Kensington Park Gardens.
- 1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Lib. Com.* 1886-8. *Trans.* 4.
- 1883 THURSFIELD, THOMAS WILLIAM, M.D., Physician to the Warneford and South Warwickshire General Hospital; 26, The Parade, Leamington.

*Elected*

- 1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity ; 27, Seymour street, Portman square. *Referee*, 1874-81.
- 1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.
- 1872 TOMES, CHARLES SISSMORE, M.A., F.R.S., 37, Cavendish square. C. 1887. *Lib. Com.* 1879.
- 1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.
- 1882 TOOTH, HOWARD HENRY, M.D., Assistant Medical Tutor St. Bartholomew's Hospital ; 34, Harley street, Cavendish square.
- 1871 \*TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.
- 1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital ; 6, Wimpole street, Cavendish square. *Trans.* 5.
- 1881 \*TREVES, WILLIAM KNIGHT, Surgeon to the National Hospital for Scrofula ; 31, Dalby square, Cliftonville, Margate.
- 1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards ; 4, St. Peter's terrace, York.
- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household ; 23, Old Burlington street.
- 1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the North-Eastern Hospital for Children, and to the London Hospital ; 15, Finsbury square.
- 1873 TURNER, GEORGE BROWN, M.D., Vernon House, Ryde, Isle of Wight.
- 1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich ; Assistant Surgeon to, and Joint Lecturer on Practical Surgery at, St. George's Hospital ; 49, Green street, Park lane.
- 1888 TYLDEN, HENRY JOHN, M.B., 38, Harewood square.
- 1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary ; 10, Langhorne Gardens, Folkestone.

*Elected*

- 1876 **VENN, ALBERT JOHN, M.D.**, Obstetric Physician to the Metropolitan Free Hospital; Physician to the Victoria Hospital for Children, Chelsea; and Assistant Physician for the Diseases of Women, West London Hospital; 27, George street, Hanover square.
- 1870 **VENNING, EDGCOMBE**, 30, Cadogan place.
- 1865 **VERNON, BOWATER JOHN**, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.
- 1867 **VINTRAS, ACHILLE, M.D.**, Physician to the French Embassy, and to the French Hospital, Leicester place; 19A, Hanover square.
- 1828 **VULPES, BENEDETTO, M.D.**, Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.
- 1854 **WADDINGTON, EDWARD**, Hamilton, Auckland, New Zealand.
- 1870 **WADHAM, WILLIAM, M.D.**, Physician to St. George's Hospital; 14, Park lane.
- 1886 **WAINEWRIGHT, BENJAMIN, M.B., C.M.**, 6, Harley street, Cavendish square.
- 1864 **WAITE, CHARLES DERBY, M.B.**, Consulting Physician to the Westminster General Dispensary; 3, Old Burlington street.
- 1884 **WAKLEY, Thomas, jun.**, 96, Redcliffe Gardens.
- 1868 **\*WALKER, ROBERT**, Honorary Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.
- 1887 **WALLACE, EDWARD JAMES, M.D.**, Holmbush, Grove road, Southsea.
- 1883 **WALLER, AUGUSTUS, M.D.**, Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road.
- 1888 **WALLIS, FREDERICK CHARLES, M.B., B.C.**, 18, St. James's street.
- 1867 **WALLIS, GEORGE**, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

*Elected*

- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. C. 1888. *Lib. Com.* 1882-5. *Trans.* 5.
- 1852 †WALSHE, WALTER HAYLE, M.D., LL.D. Edin., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption and to University College Hospital; 41, Hyde Park square. C. 1872. *Trans.* 1.
- 1883 \*WALTERS, JAMES HOPKINS, Senior Assistant Surgeon to the Royal Berkshire Hospital; 15, Friar street, Reading.
- 1851 †WALTON, HAYNES, Consulting Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.
- 1886 WARD, ALLAN OGIER, M.D., Casita, Northumberland Park, Tottenham.
- 1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.
- 1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.
- 1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton Gardens, Maida vale. *Referee*, 1881-5.
- 1877 WARNER, FRANCIS, M.D., Assistant Physician and Lecturer on Botany to the London Hospital; 24, Harley street, Cavendish square. *Trans.* 1.
- 1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary; 69, Bedford street, Liverpool. *Trans.* 3.
- 1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.
- 1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.
- 1879 DE WATTEVILLE, ARMAND, M.A., M.D., B.Sc., Physician in Charge of the Electro-therapeutical Department at St. Mary's Hospital; 30, Welbeck street, Cavendish square.

*Elected*

- 1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.
- 1840 WEBB, WILLIAM WOODHAM, M.D., Neuilly-sur-Seine, France.
- 1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.
- 1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.
- 1878 WEISS, HUBERT FOVEAUX, Assistant Surgeon to the West London Hospital; 11, Hanover square.
- 1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.
- 1854 †WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Corresponding Member, "Académie de Médecine," Paris; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 14. *Pro.* 1.
- 1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 55, Harley street, Cavendish square. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.
- 1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, and to the Royal Free Hospital; 15, Wimpole street, Cavendish square. *Trans.* 3.
- 1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.
- 1881 WHARRY, ROBERT, M.D., Physician to the Westminster Dispensary; 6, Gordon square.
- 1878 WHARTON, HENRY THORNTON, M.A., Honorary Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

*Elected*

- 1828 **WHATLEY, JOHN, M.D.**
- 1875 **WHIPHAM, THOMAS TILLYER, M.B.,** Physician to, and Lecturer on Pathology and Practical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.
- 1849 **WHITE, JOHN.**
- 1881 **WHITE, WILLIAM HALE, M.D.,** Senior Assistant Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888. *Trans.* 2.
- 1881 **\*WHITEHEAD, WALTER, F.R.S. Ed.,** Surgeon to the Manchester Royal Infirmary; Senior Surgeon to the Manchester and Salford Lock and Skin Hospital; 24, St. Ann's square, Manchester. *Trans.* 1.
- 1885 **\*WHITLA, WILLIAM, M.D.,** Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.
- 1877 **WHITMORE, WILLIAM TICKLE,** Surgeon to the Westminster General Dispensary; 7, Arlington street, Piccadilly.
- 1852 **WIBLIN, JOHN, M.D.,** Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.
- 1870 **\*WILKIN, JOHN F., M.D., M.C.,** The Warren, Beckenham park, Kent.
- 1883 **\*WILKINSON, THOMAS MARSHALL,** Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 7, Lindum road, Lincoln.
- 1837 **WILKS, GEORGE AUGUSTUS FREDERICK, M.D.,** Stanbury, Torquay.
- 1863 **WILKS, SAMUEL, M.D., LL.D., F.R.S.,** Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught, and to H.R.H. the Duke of Edinburgh; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

*Elected*

- 1883 \*WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.
- 1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital ; Surgeon to St. Luke's Hospital ; 36, Wimpole street, Cavendish square. C. 1880-81. *Referee*, 1882-8. *Trans.* 2.
- 1887 WILLETT, EDGAR WILLIAM, M.B., 60, Welbeck street, Cavendish square.
- 1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.
- 1888 WILLIAMS, CAMPBELL, 62, Welbeck street, Cavendish square.
- 1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen ; Consulting Physician to the Hospital for Consumption, Brompton [2, Upper Brook street, Grosvenor square] ; Villa de Rocher, Cannes. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.
- 1859 \*WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital ; 48, Prince of Wales road, Norwich.
- 1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton ; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888. *Lib. Com.* 1880-3. *Trans.* 5.
- 1881 WILLIAMS, DAWSON, M.D., Assistant Physician to the East London Hospital for Children ; 25, Old Burlington street.
- 1872 WILLIAMS, JOHN, M.D., Physician Accoucheur to H.R.H. the Princess Beatrice ; Professor of Midwifery, University College, London ; Obstetric Physician to University College Hospital ; 11, Queen Anne street, Cavendish square. *Referee*, 1878-88. *Lib. Com.* 1876-82.
- 1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy ; 13, Gloucester street, Warwick square.
- 1887 WILSON, ARTHUR HERVEY, M.D., 504, Broadway, Boston, U.S.A.
- 1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.



*Elected*

- 1850 \*WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.
- 1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah Hill, Upper Norwood.
- 1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.
- 1885 WOLFENDEN, RICHARD NORRIS, M.D., Assistant Physician to the North-West London Hospital; 19, Upper Wimpole street.
- 1851 †WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-88. *Lib. Com.* 1866. *Trans.* 3.
- 1887 WOOD, THOMAS OUTTERSON, M.D., 40, Margaret street, Cavendish square.
- 1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.
- 1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., Rockhampton, Queensland.
- 1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

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[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of September in each year, in order that the List may be made as correct as possible.]

# LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1833	Thomas A. Barker, M.D.	1847	W. H. O. Sankey, M.D.
1835	Thomas A. Nelson, M.D.		George Johnson, M.D., F.R.S.
1836	Alexander Shaw.	1848	Sir Edward H. Sieveking, M.D.
1838	Charles Hawkins.		Edward Ballard, M.D.
	Henry Spencer Smith.		William Wood, M.D.
1839	T. Graham Balfour, M.D., F.R.S.		Thomas Hawksley, M.D.
	Fred. Le Gros Clark, F.R.S.		Edward John Tilt, M.D.
	James Dixon.		John Clarke, M.D.
1840	Chas. J. B. Williams, M.D., F.R.S.		John Gregory Forbes.
	Samuel A. Lane.	1849	Hugh J. Sanderson, M.D.
	Sir James Paget, Bt., F.R.S.		C. H. F. Routh, M.D.
1841	Sir Henry A. Pitman, M.D.		Edmund L. Birkett, M.D.
	Sir William Bowman, Bart., F.R.S.		George T. Fincham, M.D.
	Paul Jackson.		Sir William W. Gull, Bt., M.D., F.R.S.
1842	Charles West, M.D.	1850	Richard Quain, M.D., F.R.S.
	Sir John Simon, K.C.B., F.R.S.		George Roper, M.D.
	John Erichsen, F.R.S.	1851	Sir Wm Jenner, Bt., M.D., F.R.S.
	Sir Oscar M. P. Clayton, C.B.		H. Haynes Walton.
1843	Sir Prescott G. Hewett, Bt., F.R.S.		John Birkett.
	Henry Lee.		John A. Kingdon.
	Luther Holden.		Peter Y. Gowlland.
	Edward Newton.		John Marshall, F.R.S.
1844	William Wegg, M.D.		John Wood, F.R.S.
	Thomas King Chambers, M.D.		Bernard E. Brodhurst.
	Edwin Humby.		Robert J. Spitta, M.D.
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**REGULATIONS relative to the publication of the ' Proceedings  
of the Society.'**

**That the ' Proceedings ' will be issued after each Meeting.**

**That a Copy of the ' Proceedings ' will be sent, postage free, to every Fellow of the Society who expresses a wish to receive them.**

**That the ' Proceedings of the Society ' may be obtained by non-members at the Society's House, 53, Berners Street, on prepayment of an annual subscription of five shillings and eightpence, which may be transmitted either by post-office order or in postage-stamps;—this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent carriage free, through a bookseller, or by post, the receiver paying the foreign charges.**

**That a notice of every paper will appear in the ' Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the ' Proceedings ' *only*, or in the ' Proceedings ' and ' Transactions; ' and in all cases they will be expected to furnish an Abstract of the communication.**

**The Abstracts of the papers read will be furnished to the Journals as heretofore.**



## ADVERTISEMENT.

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THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

**ADDRESS**  
**OF**  
**GEORGE DAVID POLLOCK, F.R.C.S.,**  
**PRESIDENT,**  
**AT THE**  
**ANNUAL MEETING, MARCH 1st, 1888.**

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GENTLEMEN,—The Annual General Meeting of our Society brings with it to your President the sad duty of having to speak of those whom death has removed from us since we met here on the 1st of last March.

Sixteen of our members have died during the past twelve months. Ten of these were Resident Fellows, and among them were some who were loved and revered by all who knew them, men who were highly esteemed for their work and eminent professional character. It may appear invidious to select one more than another, but the honoured names of Burrows, Arthur Farre, and Wilson Fox must long be remembered as bright examples of high professional worth.

Of the remaining six, five were Non-Resident Fellows, and one a distinguished Foreign Honorary Fellow, Bernard von Langenbeck, of whom more hereafter.

The first death that occurred after our last Annual

Meeting in 1887 was that of *Mr. George Bacon Sweeting*, of King's Lynn, in the County of Norfolk.

Mr. Sweeting was born at Bridport, in the County of Dorset, on the 26th of March, 1824. His father was a surgeon, and, as far as I can gather, practised in that town.

Mr. Sweeting was elected a Fellow of this Society in 1861. His professional education was commenced and completed at University College and Hospital. He became a Member of the Royal College of Surgeons in 1848; a Licentiate of the Royal College of Physicians, and of the Society of Apothecaries in 1851. His commencement in practice was in connection with Mr. William Image, of Bury St. Edmund's, one well known to many of us, being himself a Fellow, and a contributor to our 'Transactions.'

Mr. Sweeting remained with Mr. Image for a few years, but finally settled in practice at Lynn in 1855. Here he soon became known and appreciated for his professional worth, and succeeded in establishing for himself a high professional position, a position which he retained till a serious illness prostrated him, and no longer permitted him to work. For more than thirty years he practised in Lynn; during this time he not only was well known and trusted by the residents of the town, but acquired a considerable reputation in the surrounding neighbourhood. He was greatly appreciated by all, rich and poor, and during the whole of his professional career enjoyed a widespread reputation.

For some little time Mr. Sweeting was attached to the West Norfolk and Lynn Hospital, but in consequence of his largely increasing practice, he found himself unable to devote such time as the duties of a Surgeon to such an Institute necessarily require, and so some few years after he resigned his appointment, and then was nominated Consulting Surgeon to the Hospital.

His last illness was of short duration, he may be said to have died in harness. Some few weeks previous to his death he had been recommended rest from all work, and change of air, and consequently he left Lynn, and went

to reside with relations at some little distance from his home. But his condition did not improve; he died on the 25th of March, 1887, in the sixty-third year of his age, greatly regretted by all who knew him.

*Dr. Charles Hutton* died after a few days' illness on the 27th of March, 1887, at his residence in Lowndes Street, Belgrave Square, in the sixty-seventh year of his age. He was the last surviving son of the late Mr. William Hutton, of Beetham and Overthwaite, in the County of Westmoreland, an old patrimonial estate which had descended from father to son over a period of 400 years.

Dr. Hutton commenced and completed his medical studies at St. George's Hospital. He became a Doctor of Medicine of St. Andrews in 1845, and a Member of the Royal College of Physicians of London that same year. He studied for some time in Paris, before settling down in practice. He commenced his professional life with a small patrimony, but this was sacrificed under circumstances which only proved the unselfishness of his character, and the kindness of his disposition.

Dr. Hutton was Physician to the General Lying-in Hospital in Lambeth for over a period of twenty-seven years. He had previously been connected, as Physician, with the Hospital for Women and Children in the Waterloo Road. In connection with these appointments it was only natural that he should have devoted himself to the department of midwifery, and the disorders of women and children, and for some years he might be considered to have succeeded in practice. I have not been able to ascertain that he ever contributed to the literature of his department of practice, with the practical details of which he was most thoroughly well acquainted; he might truly be termed a good bed-side practitioner, and ready and capable to deal with all the difficulties conspicuous in the details of midwifery practice.

Only those who knew Dr. Hutton, as did the writer of this memoir, could appreciate his kindness and generosity of heart in all his associations, actions, and thoughts. He

was most genial in character, courteous to all with whom he came in contact, most attentive, we might almost say too anxious, in all that related to the interests of his patients. Professionally, his conduct was always guided by the highest principles of right and honour. His life was one of industry, self-denial, and probity, but, notwithstanding his excellent qualities and conduct, his ever most gentlemanlike and high professional character, his professional gains were not equal to his professional worth. He was a man of good education, good family, taste and high honour, and had secured the friendship of many members of the profession, and the regard of all who knew him ; simple and gentle in his manner, he gained the esteem and confidence of all who consulted him. He was taken ill on the night of the 23rd of March, and died on the 27th of that month. He was attended by his old friend Dr. Dickinson, Physician to St. George's Hospital, who tells me that Dr. Hutton had been long ailing. He was called out in the night, which was very cold, and he was almost immediately seized with an acute aggravation of his previous bronchitis, and rapidly sank under this attack.

*Dr. Daniel Wane*, late of Grafton Street, Berkeley Square, was elected a Fellow of this Society in 1852. He died on the 5th of April, 1887, in the seventy-third year of his age.

Dr. Wane was born at Penrith, in the County of Cumberland, in 1814. He was the youngest son of a large family, and suffered under the disadvantage of losing his father when but a child. He received his education at the grammar school of his native town. In the year 1832, when twenty years of age, he commenced his medical studies at Edinburgh, then famous for its many celebrated teachers, among whom were conspicuous the names of Lizars, Abercrombie, and Alison.

Dr. Wane subsequently became one of the Resident House Physicians at the Royal Infirmary, and afterwards acted as House Surgeon to Mr. Syme. In 1839 he gra-

duated ; and went to Paris, where he studied in the hospitals and schools. On his return to England he settled at Putney in general practice, where he continued till 1851, when he removed to Grafton Street, and commenced practice as a physician.

In private life Dr. Wane was much beloved. One who knew him well, writes of him to me as "a good husband and father, a steadfast friend, and, above all, an earnest Christian."

He was for many years Physician to the Blenheim Street Dispensary ; and though much occupied in practice, he does not appear to have contributed to medical literature. His health began to fail in 1882, when he decided to retire from practice. He died on his seventy-third birthday.

*Dr. William Chapman Begley*, of St. Peter's Square, Hammersmith, died on the 12th of April, 1887, in his eighty-fifth year. He was elected a Fellow of the Society in 1858, and served on the Council in the years 1877 and 1878. He was born in 1803, on the 1st of August, at Dun Cormac, New Ross, County Wexford. The early loss of his father, and the greatly reduced circumstances of his family, threw upon him the care of a mother and two sisters, until their recent deaths. Dr. Begley studied medicine in Dublin, and was a Master of Arts, and Doctor of Medicine of Trinity College.

He, together with Mr. Gaskell, afterwards a Commissioner in Lunacy, took charge of a temporary hospital for the reception of cholera patients in Manchester. He afterwards had medical charge of the patients of a large cotton factory in Derbyshire, and there he remained until the factory was burnt down, and the owner ruined by his losses. It was while living here that Dr. Begley had a large field for surgical practice, and is said to have availed himself most successfully of the opportunities afforded him. After the calamitous fire just referred to, Dr. Begley was compelled to seek some new field of employment. Soon after the opening of Hanwell Asylum he was elected Junior Assistant to Dr., afterwards

Sir William Ellis. Dr. Ellis had been selected as Chief Medical Superintendent of Hanwell, in consequence of his well-known experience and humane treatment of the insane at the Wakefield Lunatic Infirmary. Here the patients had been employed in the simple routine of gardening, agriculture, and other ordinary occupations or mechanical pursuits, which in no small measure lead to the non-restraint treatment of the insane, where it was then thought practicable, and no doubt ultimately to its general adoption.

On the retirement of Dr. Conolly from Hanwell, and the division of the work into the separate supervision of a male and a female department, Dr. Begley was placed in charge of the male department; and this appointment he retained until his retirement after thirty-four years' residence in the establishment.

During this long period of honorable service his utmost endeavours were exerted to advance the usefulness of his position, and add to the improvements of this great and benevolent institution; he had the gratification to witness the continued advance in the occupation and cultivation of the best dormant qualities of those capable of improvement, under such circumstances as may be now witnessed in all asylums for the insane.

Dr. Begley was of a studious, unobtrusive and benevolent disposition, a disposition which made him ever ready to assist and comfort the suffering, or to encourage and give hope to the weary and depressed. If evidence of these excellent qualities were requisite, I need only quote from a letter received from his sorrowing widow, which tells in simple but touching words the character of the man. "He always looked upon and spoke with feeling and great pleasure of a print in his library of 'Esquirol,' contemplating the bust of 'Pinell,' whom he thought to be the first to strike off the shackles from the insane in France. The engraving was a present to Dr. Begley from a relation of Esquirol's, and the subject he thought should be of interest to all psychologists. It represents a patient un-

fettered who, when he saw the light of day, clasped his hands and exclaimed, ' Oh, God ! how glorious ! ' ”

Dr. Begley was invited to write his experiences of Hanwell ; but his health failed, and unfortunately he was never able to place on record the results of his extensive observation of mental disorders, or the diseases which are associated with lunacy.

Dr. Begley was endued with a mind of the most active and useful benevolence. All that he left behind him, after the death of his widow, goes to establish studentships in medicine in Trinity College, Dublin ; to which college he felt he owed much, at which few such endowments exist, and where students are not unfrequently crippled as to means, as he, indeed, could say with truth had been his personal experience.

*Dr. Alfred Meadows*, well known as a physician accoucheur, died on the 18th of April, 1887, after a short illness, in his fifty-fifth year.

He was born at Ipswich. He commenced his professional life at King's College Medical School, and he is reported to have distinguished himself in a very short time by his industry and energy in the work of his student life.

His early tastes were evidently towards the study of midwifery. When qualified he was selected for the office of Resident Obstetrician at King's College Hospital, and while holding that appointment he discharged the duties which devolved upon him with much satisfaction to those under whom he officiated—the forerunner of his success in securing for himself in after life a prominent position in that department of practice.

As a student Dr. Meadows gave evidence of considerable literary taste and ability, and it was while he was still at work at the Hospital that he was appointed to edit the ' Transactions ' of King's College Medical School.

In 1857 he took his degree of B.M. of the University of London, and in 1858 he obtained his M.D. degree. In 1873 he was elected a F.R.C.P.



Soon after he had commenced practice in London he became connected with the St. George's and St. James's Dispensary as Physician Accoucheur. He was also appointed Assistant Physician for Diseases of Women and Children at King's College Hospital. For some time also he held the appointment of Physician Accoucheur to the General Lying-in Hospital.

When a vacancy occurred in the office of Physician Accoucheur to St. Mary's Hospital by the resignation of Dr. Tyler Smith, Dr. Meadows was elected without any opposition, and he retained that appointment to the period of his death.

He was the author of a "Manual of Midwifery," and of a small handy work entitled 'The Prescriber's Pharmacopœia.' He was also for a time editor of 'The London Medical Review,' well known to the profession at the time, and which for some years met with a fair amount of success.

Dr. Meadows was an active and energetic man, and he was able to bring to the contested field of practice those qualities which, combined with activity and energy, ensured him a large amount of success practically. He was much esteemed by those who knew him intimately, and much trusted by those who consulted him. He was kind and hospitable in all his social arrangements, a good mechanic, clever in the adoption of means to an end, and skilful in the manipulative details of his department of practice.

The large concourse of relations and friends assembled at his funeral near his country residence at Colnbrook, told more truly than I can express in words how great was the esteem in which he was held.

*Dr. Wilson Fox* was on his journey to his country residence, near Windermere, in Westmoreland, when he was taken ill at Preston, and there, within a few days, died from the effects of an acute attack of pneumonia, complicated with old cardiac mischief.

He had left London apparently in good health, but it

was evident to those who knew him intimately that he needed rest and relaxation from work.

The news of his rapidly fatal attack at a comparatively early age was received with sorrowful regret by the profession generally, and by all who had the privilege of his friendship. It is not too much to say that in his death Medicine lost one of her most industrious and one of her most honest workers, and the profession one of its brightest examples and one of its most honorable members.

Dr. Wilson Fox was born at Wellington, in Somerset. He came of a Quaker family in the West of England, and was connected with many other well-known families of the Society of Friends. Wilson Fox himself, in later years, joined the Church of England.

He commenced his medical studies at University College in 1850, and as student there passed through a distinguished career. He became a Bachelor of Medicine in 1854, and in 1855 took his degree of Doctor of Medicine. Subsequent to the conclusion of the usual period of student life, he was appointed House Physician to University College Hospital. On the completion of his term of office he proceeded to Edinburgh, and there held the appointment of Resident House Physician in the Royal Infirmary. Having served this office for the usual time, he quitted Edinburgh and travelled on the Continent. He passed some time at Berlin, Vienna, and other schools of medicine, availing himself of the advantages offered by the teaching of Virchow, Kölliker, and others.

On his return to England he settled in practice at Newcastle-under-Lyne. Here he was soon appointed Physician to the North Staffordshire Infirmary, and here also he soon acquired a considerable private practice. But the state of his health was not satisfactory, and after a time he decided to quit Newcastle. He came to London, and placed himself under the care of those in whom he had confidence. His health in time became quite restored, and then came the decision to make London his future

field of practice. In 1861 he was appointed Assistant Physician to the Hospital of his old School, University College; and at the same date he succeeded his former teacher, Sir William Jenner, as Professor of Pathological Anatomy.

In 1866 he was elected a Fellow of the Royal College of Physicians of London, and a few years subsequently became a Fellow of the Royal Society.

In 1867 he resigned the Chair of Pathology and succeeded to the Holme Professorship of Clinical Medicine, and also to the office of Physician to the Hospital. The duties of these appointments he continued to discharge to the time of his death.

From his earliest connection with the medical profession, Dr. Wilson Fox was eminently known as a thoroughly sincere and conscientious worker, independent in his views and clear in his deductions. He had succeeded largely in private practice, and his time was greatly occupied by increasing professional work. In addition to this constant strain, most leisure moments were occupied in the preparation of his great work, to which allusion will be hereafter made. He was tired and somewhat overdone by work. He had arranged to leave town on a certain day, but postponed his departure for twenty-four hours, that he might be present at an interview some few friends were to have the following morning early with Sir William Jenner, to request him to sit for his portrait.

On this same day, the 23rd of April, when on the point of departure for Westmoreland, he was summoned to a brother, then suffering from an acute attack of pneumonia, which proved fatal the following day; indeed, so rapidly fatal was this attack that Wilson Fox was barely in time to find his brother alive.

Wilson Fox now left Devonshire for the north with Mrs. Fox. He stopped at Preston to break the long journey to Windermere. On arrival he appeared to be suffering from fatigue and the depression natural to his

loss in the death of his brother. Early in the morning of the 28th of April he became faint, and short of breath. He was soon seen by his old friend, Sir William Roberts, of Manchester, who had hastened to attend him, and who found him suffering from pneumonia with extreme prostration. Sir William Jenner was communicated with; he at once went to Preston, and met Sir William Roberts in consultation. They found Dr. Wilson Fox extremely weak, and so ill that Sir William Jenner remained with him during the night. Dr. Askell, Dr. Wilson Fox's House Physician, was also now in constant attendance. The attack appeared to be running the ordinary course of pneumonia until the Saturday morning, when, shortly after 10 o'clock, great prostration ensued, and suddenly the breathing became much hurried, accompanied by severe pain in the lower part of the right side. His old and attached friend, Dr. Russell Reynolds, was with him soon after this prostration set in, and found that the pain was not pleuritic but muscular. The chief trouble, however, was cardiac weakness. Though some gleams of hope occasionally appeared to offer a prospect of future improvement, Wilson Fox gradually became more oppressed; in the possession of all his faculties he suddenly ceased to breathe, about 4 o'clock on the morning of the 3rd of May.

On the last day of his life he desired the following touching message to be conveyed to his pupils: "Tell my boys, the students of University College Hospital, how much I love them. Tell them that the surest road to success is to have a high standard of right and honour, and to adhere strictly to it."

Thus died one beloved by all who knew him well; whose conduct was ever influenced by the highest sense of right and honour, the great importance of which he always endeavoured, by his example, to impress on the minds of his pupils, without effort or without show,—it was an inherent part of his being.

Dr. Wilson Fox had never been a strong man, in a

mere question of health and physique. He had suffered on several occasions from attacks of illness, especially some ten or twelve years prior to his death. On that occasion he was the subject of severe pneumonia complicated by heart trouble, which left the cardiac valves in a damaged state. This latter mischief had steadily progressed to the time of his fatal illness, and when now Sir William Jenner and Dr. Russell Reynolds made their respective examinations, the valvular mischief was found to have advanced to such an extent as to render any impediment to the pulmonary circulation an almost certainly fatal complication. And so it proved, and so ended the life of one of the best of men, one of the most accurate, painstaking, clear and judicious of physicians; one whose very energy and work had largely tended to shorten, as far as it was permitted to be, a life of utility and good work.

To one who knew him, as it was my privilege to do, who was indebted to him for many professional attentions, who not only admired him for his high character and affectionate disposition, but esteemed him as a sincere friend; it is difficult to speak of such a man without a fear that what may be said of him might be thought to verge on flattery; but if I am not mistaken in my estimate of the feelings of the profession, I think I may venture with confidence to say, that those who knew Wilson Fox will acknowledge and endorse all that may be recorded of his high character and professional worth and work. I cannot do greater justice to his memory than repeat the affectionate words of one of his most intimate friends, "The sudden and premature death of Dr. Wilson Fox deprives the College of Physicians of one of its most eminent, accomplished, and beloved Fellows, and the English profession of one of its ornaments. His personal bearing and physical endowments were remarkably indicative of refinement, culture, and good breeding. Tall, spare, and erect, with features finely cut in thoughtful lines, sympathetic in expression, and eager of gaze, he produced at the first

glance the impression of intellectuality and earnest kindness which were the key to his character and the secret of his charm."

Those with whom he came in contact professionally were impressed by the simplicity of his manner, his perfect honesty in all that related to the patient as also to the consultant; his entire desire to do justice to his case; the utter absence of all conceit or assumption of superiority; his careful examination into all evidence of wrong or disease; and the quiet and patient manner in which he would listen to details, or to the opinions or suggestions of those with whom he had to consult; and his gentle, careful treatment of all, whether rich or poor, it was all the same, each appeared in his pure mind to require his every attention, and to each it was given without distinction of person or circumstance. Such was Wilson Fox in his active practical work as a physician.

His contributions to scientific medicine were numerous, and of much value to the medical world. Among them may be mentioned his researches on the development of muscular tissue, which were published in the 'Philosophical Transactions'; his observations concerning the origin and structure of cystic disease of the ovary; also his work connected with diseases of the lungs and the stomach, on both of which subjects he communicated articles to 'The System of Medicine,' edited by Dr. Russell Reynolds. He contributed three papers to our 'Transactions,' the most important of which would probably be considered that "On the Temperature, Pulse, and Respiration in Phthisis and Acute Tuberculisation of the Lung," founded on eighty cases observed by himself in University College.

But again to quote the words of the writer referred to, "The work for which he was best known was his research into the nature of tubercle; to this he devoted the best energies of his life; for many years, even when he was almost alone among pathologists in this country, he stuck manfully to the thesis that tuberculosis was a peculiar and special process, and that it was not merely ordinary chronic

inflammation, as was the popular German opinion reflected in this country, until Koch's researches were published. Dr. Wilson Fox's researches led him to believe that tuberculosis might be produced by the inoculation of indifferent material, and he expressed this opinion in a lecture delivered before the Royal College of Physicians. The publication of Koch's results, while confirming the correctness of his views, as to the special characteristics of the tubercular process, necessitated a remodelling of his views as to the etiology of the disease.

"It was owing to this that the publication of his great work on diseases of the lungs, at which he had worked with extraordinary industry for many years, was postponed. We have reason to hope that the enormous mass of materials which he had brought together, and the large collection of drawings which had been prepared to illustrate it, may soon be presented to the profession under the supervision of one well capable of doing justice to such a work."

In private practice few could have been more successful than Wilson Fox. He was early associated professionally with the Court. In 1870 he was appointed Physician Extraordinary to the Queen, and subsequently Physician in Ordinary. The latter appointment only terminated with his death. As Physician in Ordinary to Her Majesty he won the confidence and regard of his Sovereign. After his death Her Majesty was graciously pleased to have placed on record in the official Court Circular that "The Queen was much grieved to receive yesterday the news of the death, after a few days' illness, of Dr. Wilson Fox, one of Her Majesty's Physicians in Ordinary. Dr. Wilson Fox had formerly been in frequent attendance on the Queen in Scotland, and Her Majesty had a great regard for him. The medical profession loses in him one of its most distinguished members."

Such words mark the high estimation in which Dr. Wilson Fox's character and professional services were

held by Her Majesty, while at the same time they do honour to a Queen who has ever been mindful of the services rendered Her Majesty by Her Majesty's Medical Staff.

Wilson Fox was buried in a family grave at Taunton. Dr. Russell Reynolds was present on the occasion, having received Her Majesty's commands to represent Her Majesty at the funeral. He was also entrusted with a wreath to be placed on the coffin on Her Majesty's behalf; attached to the wreath was a card, on which Her Majesty had written the following words: "A mark of sincere regard and esteem from Victoria R.," an additional token of Her Majesty's sympathy and respect for one who well deserved the trust placed in him by the Queen.

*Mr. William Edward Crowfoot* was elected a Fellow of this Society in 1849. He died, after a short illness, at Beccles, in Suffolk, on May 12th, 1887.

Mr. Crowfoot was born in 1806. He commenced his medical life at Guy's Hospital, and in 1828 became a Member of the Royal College of Surgeons of England. In the same year he also became a Licentiate of the Society of Apothecaries, and in 1845 he became a Fellow of the College of Surgeons by examination.

Mr. Crowfoot commenced practice at Beccles in partnership with his father, and there for more than fifty years he lived and worked, with unwearied and constant attention to the arduous duties of a country medical practitioner; and in the discharge of these duties he appears to have won universal respect and esteem.

Mr. Crowfoot was evidently a man of much energy and thought. Early in life he took great interest and an active part in the local affairs of the town in which he resided, especially in sanitary and general questions of improvement. He acted on the Town Council of Beccles, was one of its Aldermen, and was Mayor in the years 1852 and 1853. He was, moreover, one of the most active supporters of the local Hospital, and had for many



years held the appointment of Consulting Surgeon to that Institution.

He was appointed a Justice of the Peace for the County of Suffolk in 1871, and was a constant attendant and useful member on the bench.

From all that I can gather, Mr. Crowfoot was one of those active benevolent-minded men, whose constant aim appears to have been to do good to his fellow-creatures. He identified himself largely in movements for the education and improvement of the working classes. His kindness and generosity were duly appreciated by the poor of his neighbourhood, who, with confidence of assistance or advice, were ever accustomed to apply to him as to a friend, in whom they would find sympathy in their trouble, and help if that were desirable and deserved.

Mr. Crowfoot, though active and energetic in matters municipal and educational, was by no means unmindful of the importance attached to the work of practice, or forgetful of the interests of his profession. He had an extensive general, as also a considerable consulting practice. He was an active supporter of the hospital, and a member of what was formerly the Norfolk and Norwich Pathological Society, now merged into the Norfolk and Norwich Medico-Chirurgical Society.

He was also President of the East Anglian Branch of the British Medical Association in 1855.

Mr. Crowfoot was a man of considerable mental ability, with a mind much cultivated by reading, and European travel. "To live for others," writes a friend, "to work for others, to relieve their wants, comfort their sorrows and lighten their burdens, was his constant aim."

Such appear to have been the grand moving objects of his life. He leaves behind him a memory which will long be held in respect and esteem.

*Mr. Henry Curling* was well known to many Fellows of this Society, and was highly esteemed by all who had the privilege of his acquaintance. He died on the 17th of June, 1887, in his seventy-second year.

Mr. Curling was educated at Ramsgate, and subsequently became a pupil of the late Mr. Freeman of Spring Gardens, who was at that period extensively engaged in a leading general practice. Mr. Curling subsequently studied at the London Hospital under his grandfather, Sir William Blizard, then Surgeon to that Hospital; and here young Curling carried off many prizes.

Having qualified as Member of the College of Surgeons, and Licentiate of the Society of Apothecaries in 1835, he studied for a time in Paris, Berlin, and Vienna. In Paris, for a year, he chiefly worked in the wards with Andral and Louis, and many of their cases he reported in the Medical Gazette of that day.

After travelling through Germany and Italy he settled in practice at Ramsgate, and there soon made his mark and acquired a leading position. He was one of the founders of the Ramsgate Hospital, and was for many years Surgeon to the Sea Bathing Infirmary at Margate, to the duties of which office he was most constant and attentive.

Mr. Henry Curling became a Fellow of this Society in 1846. Though always largely engaged in practice, and with considerable experience in the treatment of every variety of strumous disease of joints at the Margate Infirmary, it is to be regretted that he did not publish any observations on this most interesting and important subject.

On his retirement from the active official duties of the Infirmary, his name continued connected with it, till his death, as Consulting Surgeon.

Mr. Curling was always courteous and kind to all with whom he came in contact. He was an excellent man of business, in proof of which statement I need only mention that while engaged in very active practice he was elected Chairman of the local Gas and Water Companies of the town. At the request of the Magistrates he was appointed a Justice of the Peace for the County of Kent, and also of the Cinque Ports, to the magisterial duties of which he devoted much time.

For the last ten years Mr. Henry Curling had retired from active practice, and usually passed some months in each year in London and on the Continent. In the commencement of last year he was apparently in good health, but towards the end of January grave symptoms of disease manifested themselves, he rapidly failed in health, and died, as already stated, in the month of June, beloved and mourned by all who knew him. Mr. Henry Curling possessed great good sense and much culture. Some years prior to his death a patient, with whom he had been on most intimate terms, bequeathed him a valuable library, which proved a great source of enjoyment and occupation to him in his hours of leisure after his retirement from practice. He was the elder brother of Mr. Thomas Blizzard Curling, who occupied this chair some few years back, and who, I am happy to say, we still number among our Fellows.

*John Beswick Perrin*, of Vernon House, Leigh, in Lancashire, died on the 13th June, 1887. He was originally a pupil of Mr. Brideoak, of that town, and subsequently pursued his professional studies at King's College Hospital and Medical School. For four years he was Senior Assistant Demonstrator of Anatomy at the College, and for the greater part of two summers acted as Assistant Curator under Mr. Flower in the Museum of the Royal College of Surgeons. In 1840 he was appointed Demonstrator of Anatomy at King's College with Professor Curnow, and in 1872 was selected as one of the Demonstrators of Practical Biology under Mr. Huxley at the Science School, South Kensington. During three sessions he gave practical courses of demonstrations in anatomy to the art students of the Royal Academy of London, and gratuitous courses in 1870-71 of lectures in physiology to the Working Men's College in Great Ormond Street. In 1872 the Senate of Owens College appointed him Tutor, and in 1873 he was appointed to the additional office of Demonstrator in the Medical School.

While resident in London he was constantly busy with

his pen. He contributed many articles to the medical press, and also to the magazines and reviews of the day. In 'Nature' he published an article "On an Additional True Rib in Man," and in the 'Medical Times and Gazette' one on "The Muscular Irregularities observed in the Dissecting Room at King's College," also on "The Affinities of the Omo-hyoid and Digastric Muscles." Several other contributions from his pen found place in the 'Dental Journal,' in the 'Proceedings of the Zoological Society,' and the 'Journal of Anatomy and Physiology.'

Mr. Perrin became a Member of the Royal College of Surgeons in 1866, was elected a Fellow of this Society in 1870, and was also a fellow of the Linnæan Society.

In 1874 he became a candidate for the Professorial Chair of Anatomy at Owens College. In this he was not successful, though to his credit it should be recorded that nothing could exceed the high testimonials as to his capacity for work, and his eminent qualifications for the appointment.

Mr. Perrin having failed to secure the appointment so congenial to his tastes, and for which he was in every sense admirably suited, now decided to settle in practice at Leigh. Here he soon endeared himself to a very large circle, and had ample scope for the exercise of his benevolence and generosity. He is said to have been an excellent surgeon, and it is written of him that "no one could amputate a limb or set a fracture with greater skill and dispatch—in fact, he was a born operator."

He pursued scientific investigations through the night, when sleep had been well earned by his day's work, and thus by degrees he injured his health, and shortened his valuable life, which, if spared, would probably have enabled him to do something for the advancement of his profession, and much for the benefit of suffering humanity. Dr. Curnow, who knew him well, writes to me that "he was in many ways an exceptional man." His constant efforts were to do good, not claiming credit for himself or believing himself faultless; he laughed at those who

warned him that he allowed others to pick his brains. "Knowledge," he used to say, "should be as free as air, and no one should bottle it up."

On his retirement from Owens College an address, accompanied by a timepiece, was presented to him by the students of the Manchester Royal School of Medicine, as "a unanimous testimonial of our interest in you, and our good wishes for your future prosperity and happiness," to which address were added the signatures of more than a hundred students.

Mr. Perrin prophesied some years previously that his death would be due to cancer of the liver, and such it proved. He died in his forty-third year, regretted and beloved, a man honest, generous, and without guile.

*Mr. Richard Quain* at the time of his death was one of the oldest Fellows of this Society; he was elected a Fellow in the year 1835. He died on the 15th of last September at his residence in Cavendish Square, in the eighty-seventh year of his age. He was born near Fermoy, Co. Cork, in 1800. Dr. Jones Quain, and Sir John Richard Quain, one of H.M. Judges of the Court of Queen's Bench, were his half brothers.

On the completion of his early general education at Fermoy Mr. Quain came to London, and commenced his professional work at the Aldersgate School of Medicine, where Dr. Jones Quain was already a teacher, and who subsequently was appointed Professor of Anatomy and Physiology at University College.

Mr. Richard Quain became a Member of the College of Surgeons in 1828. He first acted as Assistant to his brother at University College, but in 1832 was appointed Professor of Anatomy. He was also about this time appointed Assistant Surgeon to the Hospital then known as the "North London Hospital," but now familiarly recognised by us all as University College Hospital; his seniors were Mr. Samuel Cooper and Mr. Robert Liston. These three names constituted the Surgical Staff when that Institution was opened for the reception of patients.

Mr. Quain became Surgeon to the Hospital in 1850, and retired from that office in 1866. He was then appointed Consulting Surgeon, and Emeritus Professor of Clinical Surgery to University College.

In 1843, when the Fellowship of the Royal College of Surgeons of England was established by Royal Charter, Mr. Quain was one of those selected to be nominated a Fellow, and was one of the senior London Fellows at the time of his death.

In 1854 he was elected a Member of the Council of the College of Surgeons, and in 1855 a Member of the Court of Examiners.

In 1868 he became President of the College of Surgeons as a matter of seniority, as was then, and it may be even said is still, the custom which prevails in the election of President by the Council of the Royal College of Surgeons of England. I trust I shall not be considered to be speaking treason when I mention that this is a custom which many of the Fellows of the College think would be "more honoured in the breach than the observance."

Mr. Quain delivered the Hunterian Oration in 1869. This was chiefly devoted to the consideration of some of the defects in general education, a subject in which he was much interested. He was also the author of 'Some Observations on the Education and Examination for Degrees in Medicine,' and also 'Observations on Medical Education.'

In 1870 he represented the Council of the College of Surgeons on the General Medical Council. He was one of Her Majesty's Surgeons Extraordinary at the time of his death.

Mr. Quain published a work on 'Diseases of the Rectum;' and, later in life, some clinical lectures.

He is, however, best known by his beautifully illustrated work on 'The Anatomy of the Arteries of the Human Body.' This, it may be certainly stated, is one of the most valuable and important publications in all that relates to the surgical anatomy of the blood-

vessels. He was greatly assisted by his cousin Dr. Richard Quain in the preparation of this work, and the illustrations were furnished by Mr. Joseph Maclise, and could not be excelled for accuracy or skill.

Mr. Quain edited the fifth edition of Dr. Jones Quain's 'Elements of Anatomy,' a work which has rendered the study of the anatomy of the human body a comparatively easy matter to the younger generation of students; while its careful investigation of the question of development of the bones adds greatly to the interest of the study of the human skeleton. It is doubtful whether any better work, for accuracy in detail or clearer style of description, has ever passed through the medical publisher's hands, and it still remains one of our best standard works on human anatomy.

He was a man of liberal education, and was well read. I am told he was a cautious rather than a demonstrative surgeon; but on all matters of clinical detail he appears to have been practical, sensible, and painstaking. So was he also in all that related to the practice of his profession. Probably he was most justly described by one who said "He shone in sound clinical principles."

He suffered greatly in the latter period of his life; want of sleep and continued pain, at the advanced age of eighty-seven, proved too serious, and he sank under these troubles.

*Mr. John George French* died suddenly at his residence in St. John's Wood on the 4th December last, in his eighty-third year. He was born at Bow, in Middlesex, in October, 1804, and was the son of the Rev. William French, rector of Vange in Essex. He was educated at Christ's Hospital. Having served his apprenticeship to a medical man at Worthing, he completed his studies at Guy's Hospital. He became a Licentiate of the Society of Apothecaries in 1825, and a Member of the College of Surgeons in 1827. In 1830 he was appointed Surgeon to the St. James's Workhouse Infirmary, and for forty-two years he discharged the duties of this office with great skill and

credit, and with much kindness and attention to the large number of patients that annually came under his care.

The Infirmary and Workhouse contained between sixty and eighty beds. This large field for work was congenial to one of Mr. French's character and energy, and he availed himself of it thoroughly. He was fond of his work and ever anxious to improve whatever appeared to him capable of improvement.

In 1853 he became a Fellow of the College of Surgeons; he was elected a Fellow of this Society in 1836.

In 1831 the cholera first visited England, and when it appeared in London a cholera hospital was established in St. James's Parish, and Mr. French was appointed the Medical Officer to it.

In the terrible outbreak which occurred in 1854 it may be remembered by some present that the late Dr. John Snow first promulgated his views that cholera was conveyed by drinking impure water. Though at first Mr. French was not satisfied on this point, he ultimately became a thorough believer in the soundness of Dr. Snow's views on this subject.

Mr. French was a very painstaking and intelligent practitioner, and was known to many for his advocacy of the treatment of carbuncle by subcutaneous incisions. He published a paper on the subject in the 'London Medical Review,' and although his proposal does not appear to have gained the approval of the profession generally, Mr. French had reason to congratulate himself on its satisfactory results in his own practice. In a letter, addressed to the President of the Local Government Board, he thus refers to the subject:—"Carbuncle is one of the most curable maladies that is ever presented to the Surgeon, so far is it removed from the apprehension of any fatal result, that the disease may be instantly arrested, and the cure (?) so rapidly effected, that the patient is enabled to pursue his ordinary avocation without interruption."

He also communicated his views on this subject in a



paper to this Society, but it was not deemed eligible for publication in our 'Transactions.'

Mr. French had for some years retired from practice, probably partly owing to increasing deafness. He had retired to bed in apparent good health, but was found dead in the morning, feeble heart action probably being the final fatal cause.

*Dr. Robert Greenhalgh* died suddenly at his residence in Cavendish Square, after a long and distressing illness, in the seventieth year of his age. He commenced his medical education at the Middlesex Hospital, and subsequently studied in Vienna and Munich. He practised for many years in Bloomsbury, and always more or less devoted himself to midwifery.

When Dr. Charles West in 1861 retired from St. Bartholomew's Hospital as Physician Accoucheur and Lecturer on Diseases of Women and Children, Dr. Greenhalgh was appointed as his successor, and he is reported to have proved himself an excellent practical teacher in this his special department.

He was elected a Fellow of this Society in 1843. He became a Member of the Royal College of Surgeons in 1842, M.D. of St. Andrews in 1853, and a Member of the Royal College of Physicians of London in 1859. He served on the Council of this Society in 1871-72, and a communication of his was printed in our 'Transactions.'

In 1877 he was compelled by the state of his health to retire from his hospital appointments, and not many years later he was obliged to relinquish private practice entirely.

He had been previously associated with the City of London Lying-in Hospital. He was Consulting Physician Accoucheur to this Institution, and also to St. Bartholomew's Hospital, to the time of his death.

Dr. Greenhalgh was well known at the Obstetrical Society, and frequently entered into debate at its meetings. He was in many respects not wanting in words and arguments to enable him to hold his own against many well-

known debaters of that Society. We may certainly give him credit for his advocacy of abdominal exploration, and his encouragement of ovariectomy. He published in the 'Transactions' of the Obstetrical Society a well-considered paper "On the Comparative Merits of the Cæsarian Section and Ovariectomy in Cases of Extreme Distortion of the Pelvis."

In 1867 he was able to show at one of the meetings of the Society the uterus of a patient from whom he had removed the cervix for epithelioma of the os eighteen months before death.

Dr. Greenhalgh must be considered as one of those who early commenced to advocate the operations of abdominal section, which has largely contributed to the advancement of abdominal surgery.

He was a severe sufferer from asthma. This greatly interfered with his daily ease and professional duties, but he was ever patient under these trials and privations—ever kind and considerate to all with whom he came in contact. Latterly he became the subject of locomotor ataxy, which progressed rapidly. He died exhausted on the 30th of October, 1887.

*Dr. Arthur Farre* died on the 17th of last December. He was the fifth son of the late Dr. John Richard Farre, of Charterhouse Square, well known and highly esteemed as a Physician. The latter was one of the original promoters, with Mr. John Cunningham Saunders in 1806, of the London Infirmary for Diseases of the Eye, now the Royal Ophthalmic Hospital in Moorfields. This was the first institution of the kind in the world in point of time, but an example which has been so generally followed that it would be difficult to mention any large town in England or in Europe and the Colonies which does not now possess an Ophthalmic Hospital, or some institution for the relief of diseases of the eye.

Arthur Farre was younger brother of Dr. Frederick Farre, many years Physician to St. Bartholomew's Hospital, and more lately Treasurer of the College of Physicians.

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The younger brother Arthur was born in Charterhouse Square in 1811, in the house in which his father lived and practised for many years. He was educated at Charterhouse, and subsequently, in 1827, became a pupil at St. Bartholomew's Hospital. The following year he entered at Caius College, Cambridge. In the intervals of the University terms he studiously attended lectures and the medical practice at St. Bartholomew's; he was constant in his work in the dissecting room, at that time conducted under many difficulties which no longer interfere with the free study of anatomy in this country. Arthur Farre was soon appointed Prosector under Abernethy, and prepared subjects for the lectures on Physiology during the last course delivered by that distinguished Surgeon. Arthur Farre graduated as M.B. at Cambridge in 1833, at the head of the Medical List, and took his Doctor of Medicine degree in 1841.

During 1836-37 Dr. Farre lectured at St. Bartholomew's Hospital on Comparative Anatomy, having succeeded Richard Owen in that Chair. Preparations illustrating his observations are still contained in the museum. He also lectured on Forensic Medicine in 1838-40.

About this period Dr. Farre turned his attention to obstetrics, and gained so much reputation in this department of practice that he was appointed in 1841 Professor of Obstetric Medicine at King's College, London, and at the same time was attached to the Staff of King's College Hospital as Physician Accoucheur. This appointment he held till 1862, when on his resignation he was nominated Consulting Physician Accoucheur to the Hospital. He was succeeded by Dr. Priestley.

Dr. Farre became a Member of the College of Physicians in 1838 and was elected a Fellow in 1843, he was Censor in 1861-62, and Senior Censor in 1865. He delivered the Harveian oration in 1872.

He was appointed Examiner in Midwifery to the College of Surgeons, and held that appointment for twenty-four years. He resigned in consequence of a

difference with the Council of the College concerning the propriety of admitting women who were not Members of the College to the examination in midwifery, and was cordially supported by the simultaneous resignation of his colleagues Dr. Priestley and Dr. Robert Barnes.

Dr. Farre was appointed Physician Extraordinary to Her Majesty in 1875, and Physician Accoucheur to H.R.H. the Princess of Wales, whom he attended in all her confinements. He also attended the late Princess Louis of Hesse Darmstadt, Princess Alice of Great Britain, in her first confinement ; also Her Imperial Highness the Duchess of Edinburgh in her first confinement at Buckingham Palace in 1874, and again in 1875 at Eastwell Park. He also attended Princess Christian, Princess Helena of Great Britain, in her first confinement in 1867 at Windsor Castle, and again in 1869 at Cumberland Lodge. He attended the Princess Mary Adelaide in all her confinements, and the Princess Leinengen in her first confinement in 1863 at Osborne. These notes have been copied from Arthur Farre's private memoranda. He was never heard to boast of or even allude to the great responsibilities thrown upon him by all this professional work in connection with Royalty. Few men, perhaps, have had the honour to attend so many members of the Royal Family, and none with greater success ; but while many members of our profession have been recognised as deserving distinction, Arthur Farre's services appear to have been entirely overlooked during his lifetime. Yet no word of dissatisfaction ever passed his lips ; he rested satisfied with the knowledge that his work had been well done and honestly done.

Arthur Farre was elected a Fellow of the Royal Society in 1839, when only twenty-eight years of age.

On the death of Sir Charles Locock, in recognition of his position as leader among English obstetricians, he was made Honorary President of the Obstetrical Society. He became a Fellow of this Society in 1844. He served on the Council and as Vice-President in 1864. But notwith-

standing his large hospital and private practice in the disorders of women and obstetrics, he failed to offer us any communication relating to his department of medicine, which all must regret, as Arthur Farre was a most observant and clear-headed practitioner.

In 1851-52 he was President of the Microscopical Society, which Society he assisted in founding; was the first Honorary Secretary, served several times on its Council, and secured for it, through the influence of the Prince of Wales, its Royal Charter.

Arthur Farre made his mark early in life as a scientific worker as well as a thoroughly good practical physician. The article "Uterus and its Appendages," with numerous illustrations from his own original drawings, published in the 'Cyclopædia of Anatomy and Physiology' in 1868, of which it forms two complete numbers, was regarded as the standard authority on that subject, and may truly be said to be a model of scientific precision, both in its anatomical and physiological details. It is a compendium of all that relates to the anatomy, physiology, and pathology of the uterus, together with the changes which the uterus undergoes during pregnancy.

He communicated many papers to the Royal and Microscopical Societies; and a paper published in the 'Medical Gazette' in 1835, on the *Trichina spiralis* (then recently discovered), illustrated by woodcuts, thus showing its minute anatomy and pointing out the true structure of this parasite. He was also the author of "Observations on the Minute Structure of some of the Higher Forms of Polypi." "Lectures on Comparative Anatomy," reprinted from the 'Philosophical Transactions.' "An Account of the Dissection of the Human Embryo, with Observations on the Early Development of the Human Heart," and other interesting papers.

Dr. Farre had been much over-worked for some time prior to his retirement from practice. The loss of a beloved wife came as a terrible blow to his happiness, and much saddened his later days. Then, in the midst of

much professional work and consequent mental strain, occurred a sad accident by which he sustained a compound fracture of his ankle-joint. For many weeks it was doubtful what the result might be, but gradually the wound healed, and in the course of time he was able to move about on crutches. But though he was in time able to discard these for a stout stick, he never was able to use the injured limb without discomfort and pain. He retired from all professional work, and passed his latter days in his favourite amusements of music, literature, and occasional social meetings with some few friends; latterly these meetings became less frequent owing to failing strength and increasing pain on movement.

Independent of Arthur Farre's scientific work and professional excellence, it is only due to his memory to add there was all that in him that represented the perfect gentleman. Combined with the highest sense of honour in regard to all that related to his department of medicine, he was a model physician accoucheur, guided by the highest principles, and never influenced by greed. He worked for the good of mankind and his reward was the high estimation in which he was held by his college and the profession generally, and the sincere and warm friendships he secured in private. He was not a man of any public demonstration, but those alone who knew him intimately could sufficiently appreciate his kind, gentle, and generous nature. A man of many genial qualities, and much wit and sharpness of intellect, of most tender feelings, and ever ready to do a kindness to a professional brother who might need his advice or assistance. It was my privilege, as it was always my pleasure, to have much to do with him during his long confinement to bed in consequence of his accident. I can truly say I never met with a more trustworthy, confiding, high-minded, and grateful character than that of my old and esteemed friend Arthur Farre.

*Sir George Burrows* died on the 11th of December of last year. He was born in 1801, in Bloomsbury Square,

where his father, Dr. George Mann Burrows, then resided. His early education was commenced at a school at Ealing, and in due time he went up to Cambridge, and entered at Caius College. Here he soon obtained a Tancred Studentship. But, putting aside medicine for a time, he devoted his attention more exclusively to the special study of the University, and ultimately came out tenth Wrangler in the Mathematical Tripos of 1825. He was at once elected a Fellow of his college, and Junior Mathematical Lecturer. He occupied his spare time with pupils, and is said to have been a popular and successful teacher. He subsequently left Cambridge, and commenced his professional work as a pupil at St. Bartholomew's Hospital, and there he worked under Dr. Latham and Sir William Lawrence, and served the latter as dresser for twelve months. He obtained his M.B. degree in 1826, and, having completed the time of student life, he travelled abroad, and studied in Paris and Pavia and at some of the schools in Germany. When the cholera appeared in London in 1832, he was placed in charge of an auxiliary Hospital, established by the Governors of St. Bartholomew's to meet the great demand on their accommodation. In 1834 he was appointed the first Assistant Physician to take charge of out-patients. His first appointment as Lecturer was to the Chair of Forensic Medicine, and subsequently he became joint Lecturer with Dr. Latham on Medicine.

Dr. Latham retired from St. Bartholomew's Hospital, as Physician, in 1841, in a great measure in consequence of the state of his health. Dr. Burrows was now appointed sole Lecturer, and also Physician to the Hospital, an appointment he held with great distinction till 1863. He then retired from the active staff, and was elected Consulting Physician, being the first medical officer connected with his Hospital on whom this honour had been conferred. On this occasion a testimonial was presented to him by his colleagues, in recognition of his high character and past services to the Medical School and Hospital. In 1832 Dr. Burrows became a Fellow of

the Royal College of Physicians, where in later years and at different intervals he delivered the Gulstonian, the Croonian, and Lumleian Lectures.

At the College of Physicians he occupied, at various periods, the offices of Member of Council and Censor, and he represented the College on the General Medical Council. In 1871 he was elected President of the College of Physicians by the unanimous voice of the Fellows, and in recognition of his administrative qualities and high conduct while occupying that chair he was called upon to undertake the duties of that important and honorable position for five consecutive years.

He was appointed Physician Extraordinary to Her Majesty in 1820, and on the death of Sir Henry Holland succeeded him as Physician in Ordinary to the Queen. This appointment he held till the time of his death. In 1874 Her Majesty was graciously pleased to confer on Dr. Burrows the honour of a Baronetcy—it came unsolicited, but not undeserved—and, by all who knew him, was considered to be bestowed on one well worthy of such distinction.

Sir George Burrows was always active. In his earlier days at Cambridge he was well known as a good “oar” on the river. He took a most active and useful part in all that related to the interests of his profession. He was a Member of the Senate of the University of London, for a time President of the General Medical Council, President of the British Medical Association, and in 1869–70 President of our Society. He was a Fellow of the Royal Society, D.C.L. of Oxford, LL.D. and Honorary Fellow of Caius College, Cambridge. He had long been a liberal supporter of the Society for the Relief of Widows and Orphans of Medical Men, and till within a short period of his death had held the office of President, and most regularly attended the Committee Meetings.

Sir George Burrows was not a voluminous writer, but what he wrote was the result of his own honest observation and opinion, and as such valuable and instructive.



He was the author of a work on 'Disorders of the Cerebral Circulation,' and one on the 'Connection between affections of the Brain and Diseases of the Heart,' being the substance of his Lumleian Lectures delivered in 1843-44. He also contributed several articles to 'Tweedie's Library of Medicine.'

He was a man of excellent business habits, conscientious in the discharge of every duty, large-hearted and benevolent, ever actuated in all his thoughts and actions by a high sense of honour and justice. Sir James Paget says of him that his lectures were "plain, judicious, and complete; all that was generally accepted or most probable, all the best facts that he could learn in practice, or in a fair range of reading, he explained simply and in good English; he seldom suggested or discussed hypotheses; he was never dull, or cold, or trivial, but he never seemed to wish to be thought brilliant."<sup>1</sup> It was only necessary to meet him in consultation to be satisfied of his carefulness in the examination of a case, of his judgment in weighing the importance of every symptom, and of the general correctness of the opinion arrived at, added to the extreme simplicity of treatment and the common-sense advice he gave his patient. Though to some he may have appeared, in consultation, somewhat reserved in manner, and not too lavish of words, to those who knew him intimately he was always genial, tender, and considerate, ever ready to do any act of attention to a sick professional brother, and large-minded enough and willing to bring to notice the services of any member of the profession whom he considered worthy of recognition and reward. It is a personal gratification to me to be able to record my estimate of his high professional character, of his undeviating right and exemplary professional conduct, and of his constant endeavour to maintain the dignity of his order. I had much to thank him for. No one could have been more attentive or more feeling than

<sup>1</sup> "Memoir of Sir G. Burrows," by Sir James Paget. 'St. Bartholomew's Hospital Reports,' 1888.

when, on more than one occasion, I was compelled by illness in my family to seek his professional aid.

Shortly before his death Sir George Burrows tendered his resignation as trustee of this Society, in consequence of advancing years. This office he had held for many years. The Council, in accepting his resignation, desired me as your President to communicate with Sir George; and while acknowledging the receipt of his letter, to convey to him the thanks of the Council for his long services; and with your permission I will read that answer, as it entirely alludes to those services. "In accepting your resignation the Council desire me to convey to you their grateful thanks for your numerous and long services to the Society as trustee for many years; as Member of Council, and as Vice-President and President. It has fallen to the lot of few to have thus honorably served the Society, it is not the lot of many to have served it so faithfully, so efficiently, and with such perfect success as to ensure the general and sincere approbation of the Fellows of the Society. It is for these past services that I have now to beg of you to accept, on behalf of the Council, their unanimous and cordial thanks; thanks which we all feel the whole Society would heartily join in, were it called together to express an opinion on the subject. In the name of the Fellows, as in that of my own, I am sure I may express the sincere wish that the evening of your life may be one of prolonged peace and happiness, one of freedom from pain or suffering."

He received this letter but a few days previous to his death; he was never well enough to reply to it.

Sir George Burrows was elected a Fellow of this Society in 1833. He was the oldest Fellow on our list at the time of his death.

He married in 1834 the youngest daughter of Mr. Abernethy, one of his former teachers at St. Bartholomew's Hospital. Lady Burrows died in 1882, after a married life of great happiness, though saddened by the loss of several children.

In the autumn of 1886, while residing in the country, he met with a severe accident, a fall on his back when entering his house. He was very much shaken and suffered great pain, with considerable muscular trouble, a condition which necessitated his confinement to bed for many weeks. He returned to town, but he never appeared to have entirely shaken off the effects of this accident, it left him permanently weaker and less capable of exertion. As the summer advanced he was again able to see his friends and take carriage exercise, but to those about him the decline of power gradually became more evident, until the end. The morning previous to his death he got up, dressed, and was ready to receive Canon Capel Cure when he arrived to administer the Holy Communion. As the day went on he gradually became weaker and passed away in sleep soon after twelve the same night, in his eighty-seventh year. He was buried at Highgate. The large assembly of relations, colleagues, old pupils, friends, and even some grateful patients, present at his funeral bore best testimony to the worth of the man, and the affection with which he was regarded by all who knew him well.

*Dr. Walter Benoni Houghton* was elected a Fellow of this Society in 1878. He commenced his professional work as a student at University College and Hospital. Those who knew him best report that a more brilliant student career than that of Walter Benoni Houghton could scarcely be conceived, and that he bore the honours which fell to him with a modesty and grace which almost removed the disappointment of those whom he excelled.

Dr. Houghton was born in 1851. He was educated at Cambridge House, Blackheath. In 1866 he passed the Cambridge Local Examination with honours. The year following he passed the Oxford Local Examination, and became an Associate in Arts of that University. He matriculated at the University of London in 1868, passed the preliminary Scientific for M.B. in 1869, and the same year entered as a student at University College and Hospital. In his first session he obtained the second

Silver Medal for Chemistry, and in the second session the first Silver Medal for Anatomy and Physiology. In 1872 he obtained the Gold Medal for Surgery, and the Gold Medal for Medical Jurisprudence. In the same year the distinctions he gained at the University of London were remarkable; on passing the first M.B. he was awarded two Exhibitions, and Gold Medals for Anatomy and for Organic and Pharmaceutical Chemistry respectively.

In 1874 he took both the degree of M.B. and B.S., and also became a Member of the Royal College of Surgeons.

In 1876 he became an M.D. of London, and a Member of the Royal College of Physicians in the following year.

He acted as Physician's Assistant, and Obstetric Physician's Assistant at University College in 1877. He then soon after was appointed Medical Registrar, and Lecturer on Botany at Charing Cross Hospital, and then Assistant Physician.

A career so bright, hopeful, and promising, was, however, only too soon and too sadly cut short by severe attacks of illness, into the details of which it is not to our purpose to enter; but the result was that he had ultimately to resign his appointments at Charing Cross Hospital, and seek change of residence. He settled in St. Leonards; but the change did little or no good; he gradually lost health, and it was soon evident that serious constitutional damage was established and making sad progress. He left St. Leonards, and went some few months back to reside with a brother at Teddington. His malady, lung mischief, gradually progressed, and he succumbed on the 12th of February last.

Dr. Houghton was a man of extensive general as well as professional reading. The brightness and the warmth of his disposition are much spoken of; his kindness and generosity, and readiness to assist the poor and afflicted, if heightened by his own sufferings, were none the less inherent qualities with him. Dr. Hare, who knew him intimately, and to whom I am indebted for most of the particulars I have given of Dr. Houghton's career, has

written the following kindly lines in a memoir of him :  
“In the battle of life, so far as his health allowed, his career was one of uniform success ; and if in the battle of life he broke down and failed, the loss is ours.”

*Bernard von Langenbeck*, late Professor of Surgery in the University of Berlin, was elected a Foreign Honorary Fellow of this Society in 1856. He died on the 29th of September last, in his seventy-seventh year, from the effects of cerebral apoplexy. He studied under his uncle, the celebrated Surgeon and Anatomist at Gottingen, and here Bernard Langenbeck graduated in 1835. For a short time he practised as a Surgeon at Gottingen ; but in 1842 he was appointed Professor of Surgery at the University of Kiel, and Director of the Friedrich's Hospital, which necessitated his residing in the latter town.

In 1848 he became attached to the Army, and during the war of the Duchies against Denmark, he had the direction of the Surgical department of the Hospitals. This same year he succeeded Dieffenbach at Berlin, as Director and Professor of the Berlin Royal Surgical Clinical Department. Langenbeck was continuously employed during the great and severe wars in which Prussia was engaged against Denmark in 1864, against Austria in 1866, and lastly during the great contest with France in 1870. His experience under such circumstances could hardly be surpassed ; it may be truly said to have been immense, and great was the ability with which his work was performed, and large the results as regarded the improvements introduced into military and general surgery. He did much to advance conservative surgery by recourse to resections, he did much to introduce new methods of operating, and new operations in place of some of the old styles of treating wounds and various injuries or surgical maladies. He was a good operator, and full of resources as occasion required.

Langenbeck was Chief Surgeon on the Staff of the Emperor, and personally attached to him during the late

war carried on by Germany against France. "His name," writes Dr. von Lauer, "will never be forgotten either in the Prussian Military Medical Training Schools, or by the whole body of the Military Medical Department; but those officers of the Sanitary Corps who had the privilege of enjoying personal relations with him, will always gratefully remember the kindness of his manner and the loyalty of his friendship."

His contributions to the literature of his profession are to be found chiefly in 'Langenbeck's Archiv,' which he started, and edited for many years; a great work, which embodies most of what is surgically good in German literature during the last thirty years.

Langenbeck wished to retire from practice some few years ago, and to live at Wiesbaden, but the Emperor would not consent to this. Failing health and strength, however, compelled Langenbeck at last to quit the field of active work, and he retired to a house he had built for himself on the slope of the Nersberg.

Langenbeck had a great love for England and English Surgery. He was conspicuous amongst us in 1873, at the meeting of the British Medical Association; and again in 1881, during the meeting of the International Medical Congress. He was much loved by all who knew him, deservedly renowned as a great Surgeon, and honoured by his King and his countrymen. Such was Bernard von Langenbeck, who most highly appreciated the compliment paid him, when nominated by this Society one of our Foreign Honorary Members.

And here, gentlemen, ends the professional life-history of those whom death has removed from the muster-roll of the Royal Medical and Chirurgical Society in the year now concluded. I have endeavoured to the best of my ability to do justice to the professional work and life of each, but have avoided, as far as was consistent with my duty, details of much that relates to the private incidents of life, or matters which concerned family history more than professional character. I trust I have not erred in so doing.

To do justice to all has been my sincere desire, and in this endeavour I have been greatly assisted by the numerous relations and friends of those of whom I have spoken, and also by the various records which have appeared from time to time in the pages of the 'Lancet' and the 'British Medical Journal.' Altogether, I think the Fellows of the Royal Medical and Chirurgical Society may feel satisfied that the work done by those taken from us, some silent and without display, some more conspicuous and perhaps more original, may be looked upon as surely useful to mankind, and as assisting in the advance of Medicine.

If such is our past, let us hope more for our future ; for all here must feel that though of late years a great and good work has been accomplished in the treatment of disease, a great deal more has to be effected to enable us successfully to battle with much that tends to shorten life, or that renders that life one of prolonged suffering.

In reviewing the work that has been done by those who have contributed to the 'Transactions' of the Society during the two years I have had the honour to preside in this chair, I wish to draw your attention again to some of those papers, which occupied your attention on different occasions, and which appear to me to deserve much consideration.

I first refer to the valuable contributions of Mr. Rickman Godlee, Mr. R. W. Parker, and Mr. Haward, on the treatment of cases in which kidney complications required the assistance of the Surgeon, a branch of Surgery of such comparatively recent practical consideration that it appears to me we cannot at present have too much information brought under our notice as the result of the immediate experience of our Fellows, and I am sanguine enough to hope that much more material will yet come to us to help towards good and thorough practical results.

Then, to refer to a somewhat kindred question, I may allude with satisfaction to the interesting communications

of Mr. Rivington, Mr. Jacobson, and Mr. Barwell, on the removal of urinary calculi by the supra-pubic operation—an operation which every Surgeon must acknowledge to be a great addition to our means of saving life in cases for which any other form of operation holds out but a small, if any, prospect of benefit.

Dr. Vivian Poore's valuable communication on writers' cramp and impaired writing power is again one of great practical interest, and came as a welcome addition to his previous report on the same subject—a subject which the writer has considered with the most careful observation, and the results of which as communicated to us will form a most important addition to our 'Transactions.'

I have also to refer to Mr. Knowsley Thornton's important communication on the removal of the spleen. This is an operation which has not yet been sufficiently tested to be at present pronounced as sufficiently promising in its results—not that I wish by this remark to be considered in any degree as discouraging its adoption, but rather to point out what all know who have had experience in the treatment of this enlargement, that it is most frequently connected with a constitutional condition inimical to any surgical interference—to say nothing of the amount of blood which may be lost in its removal.

I must now allude to the interesting and suggestive communication by Mr. Malcolm on the condition and management of the intestine after abdominal section, considered in the light of physiological facts, a subject in which every Surgeon must feel deep interest, and from the perusal of which communication will find ample material for thoughtful digestion.

The interesting and successful treatment of a case of occlusion of the left bronchus by a pencil cap, communicated by Dr. Cheadle and Mr. Thomas Smith, I think will be allowed to be one of the most practically important communications we have received this last session. Such cases, as we are all aware, are by no means common, nor simple to deal with when met in practice: and the



publication of it in our 'Transactions' will be a valuable addition to the surgical literature of this subject.

The case of intraperitoneal rupture of the bladder, related by Mr. Walsham, and treated by abdominal section, with recovery, is again one of the more recent and one of the most satisfactory advances in Surgery of the present day. A similar case has been recorded by Mr. Timothy Holmes, and was the first instance, I believe, of this injury being thus successfully treated in England.

I cannot conclude this reference to some of the work of the two past sessions without drawing attention to those most interesting cases which were shown us by Mr. Arthur Barker, on the occasion of our last meeting. These were two cases in which abscess of the brain had been successfully dealt with by Mr. Barker by trephining the skull, and evacuating the abscess. Such results may truly be termed the best triumphs of modern surgery. The exactness of diagnosis, and the success of treatment were all that could be desired. Medical and physiological teaching and practical skill could not have desired a more satisfactory result. These patients were literally rescued from a certain and early death, by thoughtful consideration and care adapted to skilful and judicious treatment.

However much we may have reason to congratulate ourselves on the work of our Society, and on the advancement of many sections of medicine and surgery, we know too well how much yet remains to be done in every department of our profession—how much in the prevention, in the treatment, in the removal of disease. I avoid the term "cure" as inapplicable to the results of most, if not of all of our dealings with maladies. But there is still a large field of work for the young labourer in medicine in which an abundant harvest may be gathered by those who will apply their talents and observations to the investigation of any section of medical study. Cancer and tubercle offer abundant materials for future investigation, and will well reward the time and attention devoted to it. Is it too much to hope that some one hereafter

may be able to point to the successful and sure treatment of one or both? We hope not; but in work alone can that end be obtained. This Society has long and well encouraged work in every branch of medicine. I would ask, can it not do something more towards the encouragement of work, in respect to these formidable enemies to human life, that might tend towards arrest or removal?

It only now remains for me to mention how highly I appreciate the honour that was conferred on me when you elected me your President, and to thank you for the kindness and courtesy with which I have been ever received since I occupied this chair. I wish to take this opportunity to thank the Members of Council for their support and attention on all occasions of our meetings, and last, though not least, to express my best thanks to our Secretaries, Dr. Cheadle and Mr. Howard Marsh, for their help and cordial assistance throughout the tenure of my office.

*Note.*—Dr. Barnes informs me that the resignation of Dr. Farre and Dr. Priestley *followed* his; and that these resignations did not arise upon the question of admitting “women,” but “persons” to register upon a fragmentary examination.

*Erratum.*

Page 80, sixth line from bottom, “first” should be “second.”



THE CONDITION AND MANAGEMENT  
OF THE  
INTESTINE AFTER ABDOMINAL SECTION  
CONSIDERED IN THE LIGHT OF PHYSIOLOGICAL  
FACTS.

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A KNOWLEDGE of the nervous mechanism of the intestine is important to the comprehension of some of the phenomena which may be induced by an abdominal section. As a preliminary to the study of these phenomena, I propose, therefore, very briefly to direct attention to certain facts connected with its nervous supply which seem to me to have a direct bearing on the condition of the intestine after it has been interfered with during an operation.

The nerves of the intestine are mainly derived from the plexuses of the sympathetic which accompany the branches of the mesenteric arteries.<sup>1</sup> In the wall of the bowel these nerves terminate in two plexuses, one of which (the plexus myentericus of Auerbach) distributes its fibres to the muscular coat of the gut, while the other (Meissner's

<sup>1</sup> Turner's 'Introduction to Anatomy,' pp. 711 and 723.

plexus) gives branches "to the muscularis mucosæ, the smooth muscular fibres of the villi, and the glands of the intestine."<sup>1</sup> There is evidence that the nerves of the bowel are in intimate physiological connection with the central nervous system.<sup>2</sup> But the plexuses in the wall of the gut possess also, and in a very high degree, an automatic power comparable to that of the ganglion-cells of the heart. This automatic power enables the intestine even "when cut out of the body to execute, apparently spontaneously, movements for some time;"<sup>3</sup> and during life it plays an important part in the performance of all the functions of the gut. These functions are therefore readily influenced by local stimuli, which may be chemical, mechanical, thermal, or electric in their nature, and which may act from within the bowel or from without. Or the stimulus may consist in some condition of the blood passing through the intestinal vessels.

The effect of stimulation of Auerbach's plexus is first to increase peristalsis, which may become violent, spasmodic, and painful. But if the stimulus be sufficiently severe or prolonged, paresis or complete paralysis of the muscular power of the gut results. The condition of the blood flowing through the intestinal capillaries has a most important influence on the intestinal movements. Marked irritability, followed by paralysis of the intestine, may be induced by congestion or plugging of its vessels; and the spontaneous evacuation of the bowel which sometimes occurs on the approach of death is attributable to vascular changes in its wall. Inflammation too may be accompanied by irritability of the muscular wall of the bowel, as is frequently observed in catarrh of its mucous membrane, while severe peritonitis produces absolute muscular paralysis of the gut.<sup>4</sup>

The secretion of the digestive juices is also under the

<sup>1</sup> Stirling's translation of Landois' 'Physiology,' 2nd edit., p. 337.

<sup>2</sup> Ibid., p. 350.

<sup>3</sup> Ibid., p. 337.

<sup>4</sup> Ibid., p. 337, *et seq.*

control of the nervous system, and it has been shown that division of the nerves distributed to the gut is followed by a copious flow of watery secretions from the intestinal glands;<sup>1</sup> but neither the influence of the various nerves and ganglia on the secreting structures nor the extent to which the glands are capable of independent action is well determined. Nor is the effect of nervous conditions on the development of gases in the intestine well understood. The production of these gases has been attributed to processes of fermentation, to the exchange of gases in the intestine for those in the blood, and to excessive swallowing of air; but these processes do not seem sufficient to account for the large quantities of gas which are developed in the intestines in certain nervous diseases. Simple transfusion<sup>2</sup> and secretion<sup>3</sup> of gases by the mucous membrane have been offered as explanations, but neither has been proved to take place to any great extent.<sup>4</sup> It seems to me that an excessive development of gases in the intestine may sometimes be produced by a simple chemical action of the secretion (it may be an altered secretion) from one gland, or set of glands, on that from another. In Stirling's translation of Landois' 'Physiology' it is stated<sup>5</sup> that carbonic acid is set free in the stomach on the contact of the saliva, which contains carbonates, with the acid of the gastric juice. But the pancreatic, biliary, and intestinal secretions also contain a considerable percentage of carbonic acid in the form of carbonates.<sup>6</sup> In the gastric juice during healthy digestion "the amount of free acidity is, however, slight, because the hydrochloric acid which the gastric juice contains combines for the time being with pepsin and proteids, forming a

<sup>1</sup> Stirling's translation of Landois' 'Physiology,' 2nd edit., p. 397.

<sup>2</sup> Foster's 'Physiology,' 3rd edit., p. 278.

<sup>3</sup> 'American Journal of Medical Science,' N. S., iv, 403.

<sup>4</sup> Kirke's 'Physiology,' 3rd edit., pp. 366, 367; Quain's 'Dict. of Medicine,' art. "Flatulency."

<sup>5</sup> 2nd edit., p. 359.

<sup>6</sup> Stirling's translation of Landois' 'Physiology,' 2nd edit., pp. 361, 383, 395.

compound which does not give an acid reaction.”<sup>1</sup> But we know that urine, sweat, and saliva may be secreted profusely under the influence of purely mental stimulations; and if a flow of gastric and intestinal juices should occur from similar influences or from other disordered conditions, when the stomach is empty or contains food which is unsuitable in quantity or in quality, then the gastric secretions may pass, unaltered, into the duodenum, and may there mix with the secretions from the pancreatic, biliary, and intestinal glands. Moreover, from observations by Reichman, “gastric juice appears sometimes to be secreted with a larger proportion of acid than normal.”<sup>2</sup> A profuse flow of gastric juice, especially if thus rich in acid, in the absence of a normal object for its energies, would be expected, on mixing with the juices in the duodenum, to set free carbonic acid gas by the action of its acid on the carbonates which these juices contain. Other less understood chemical reactions may take place, and as there can be no doubt that nervous conditions do influence the amount and the quality of the secretions, it is conceivable that large quantities of gas might thus be produced almost with explosive suddenness; and this seems a possible explanation of the enormous development of flatus which takes place in certain conditions.

The exact sources of the gases in the alimentary canal, and the causes of their presence in excessive quantity, are much in need of elucidation; but there is no doubt that excessive flatulence “seems to be in some measure connected with defective or deranged innervation, for flatulent accumulations in the stomach and bowels, not attributable to any other cause, are frequent in nervous affections such as hysteria, hypochondriasis, and strong mental emotions.”<sup>3</sup>

It would appear therefore that all the functions of the intestine are under the control of the automatic nervous

<sup>1</sup> Lauder Brunton's 'Diseases of Digestion,' p. 12.

<sup>2</sup> Ibid., footnote, p. 33.

<sup>3</sup> Wood's 'Practice of Medicine,' vol. i, p. 713.

plexuses in its walls; and that these plexuses, while closely associated with, and to a great extent controlled by, the central nervous systems, are readily affected by any disturbing influences which may act directly upon them.

Many disturbing influences are brought to bear on these local nervous centres in the wall of the gut whenever the surgeon interferes with the peritoneum. The disturbance may be very slight, as in the operation of tapping the peritoneum for ascites. On the other hand the intestines may be subjected to the most abnormal conditions and to much physical injury, as in the removal of an adherent tumour from within the peritoneal cavity. Or a septic agent may be introduced. In all cases the nervous plexuses of the intestine are more or less affected, their activity being first increased and afterwards paralysed in proportion to the severity and duration of the stimulus. A condition is produced comparable to that which gives rise to the after-constipation which frequently follows the administration of castor-oil and other purgatives, and which owns exactly the same cause,—exhaustion of the nervous centres in the wall of the intestine.

Theoretically, then, paresis, or paralysis of the gut, results from every abdominal section. Practically the effect is often demonstrable. When an abdomen is first opened the presenting coils may be seen in a state of active peristalsis, but after a short exposure they become limp and motionless, and for from an hour or two to some forty-eight hours or even longer after the operation there is no sign (except the vomiting due to the anæsthetic) of any intestinal activity. Flatus does not escape from the bowel, and there is no abdominal distension. After a variable time, however, recovery of function in the gut is shown, if the case do well, by the passage of flatus from the anus, when a tube is passed through the sphincter ani; by freedom from abdominal distension and from vomiting; and, later, by the rectum becoming full of fæces or by the occurrence of an act of defæcation.

But though the paralysis does not continue, the mus-



cular power of the intestine is diminished for some time after operation, and this diminution of power is greatly aggravated by the want of exercise. The constraint which is necessarily put on the position of the patient also hampers the power of the gut by impeding the use of the accessory muscles of defæcation for the purpose of expelling flatus. The existence of temporary intestinal paresis at this time is shown by the facts that flatus may escape freely if a tube be passed through the anus, when if left to the efforts of nature it is retained by the sphincter ani; that when an obstruction does occur the intestines may, and generally do, distend without the least pain; and that the bowels almost always require some assistance by enema or by laxative medicine at their first movement.

It now and then but rarely happens that very little gas is generated in the intestine as a consequence of abdominal section; on the contrary, it is usual for a good deal to be evolved after the intestine begins to revive; and in that case, if the patient do well, flatus escapes freely every three or four hours when a tube is passed into the rectum to facilitate its exit. This free escape of flatus continues usually for three or four days after its commencement, and then the amount of gas which escapes gradually decreases until it is no longer noticeable. The amount of gas which may be generated is most obvious when for some reason it does not escape from the bowel. Under such circumstances immense abdominal distension may take place in a few hours. This excessive development of gases during the recovery of the intestine from the effects of operation seems to be "connected with defective or deranged innervation," and in this respect resembles the flatulency of hysterical and other nervous conditions.

But whatever the cause of the development of gases in the alimentary canal after abdominal section, their speedy escape, through the rectal tube, or by the efforts of nature, is one of the most important signs that a case of this kind is doing well. If flatus does not escape freely some

condition may be presumed to exist which interferes with the passage of the contents of the alimentary canal, or with the peristaltic action of the gut. If distension of the abdomen or vomiting follow, this presumption becomes a certainty, and it is then of the greatest importance to discover, and if possible to remove, the condition which causes the retention of flatus.

This may be :

1. Paralysis of the muscular wall of the gut.
2. Some malposition of a portion of the gut, brought about by the disturbance and rearrangement of the abdominal contents during operation.
3. Blocking of the lumen of the intestine by fæces.
4. A malformation, congenital or acquired, acting alone or by aggravating some other condition.
5. The accumulation of serum or of blood and serum in the peritoneal cavity, or escape into that cavity of irritating matter of any kind.
6. Inflammation of the peritoneum covering the gut.
7. General septic peritonitis.
8. Some pathological state of the bowel, such as one of the common forms of hernia, an intussusception, and others, which may occur as a concomitant disease in no way brought about by the operation, and to which therefore I will not refer again.

Of these we have seen that the first—paralysis of the muscular wall of the gut—follows every abdominal section in proportion to the amount of the interference with the intestines. The paralysed condition, if no other complication occurs, usually passes off in a few hours, but there is evidence that the bowel is distinctly wanting in power for from four to six or more days after operation. This condition is a most important factor in the production of obstruction of the intestine after abdominal section. It is obvious that in this state the bowel must be prone to yield to, rather than to remove, any opposition to the downward passage of its contents, so that a very slight impediment, which would be of no consequence whatever

during health, may lead to the most disastrous results. It would even appear that at this time such natural obstacles as exist at the anus, and in the friction of the more solid fæces which accumulate in the rectum and colon, may be the cause of a fatal obstruction.

Mr. Lawson Tait has twice seen death from paralysis of the intestine a week after operation. He says: "Two of my fatal cases, where the ligature had been used, were due to a kind of paralysis which was perfectly inexplicable. Dr. Batty tells me he has seen the same thing. In both instances the patients had made satisfactory progress until the sixth day after operation, without the slightest interruption, when suddenly the abdomen became greatly distended, incessant vomiting occurred, and the patient rapidly sank. After death nothing could be found except enormous distension of the abdomen by fluid fæces and gases. In both cases temporary relief was produced by tapping the intestines. The only explanation I can offer of the fatality in these cases is that some mysterious influence, similar perhaps to that which causes tetanus, brought about this unexpected and inexplicable end."<sup>1</sup>

I have met with a very similar case. Ovariectomy had been performed, and the patient progressed favorably until the seventh night after operation. There was then no cause for alarm when I saw her at 6.30 p.m. Vomiting came on about four hours after this, but the nurse did not send for assistance, and the patient vomited a great deal all night. In the morning, between 6 and 7 o'clock, the bowels moved spontaneously, and a large motion was then brought away by enema. Vomiting immediately ceased and did not return, but at 7 a.m. the temperature was 99.8° F.—the highest that had been recorded since the first night after operation. At 9 o'clock the temperature was 101.2°, and at the morning visit, at 9.30, the abdomen was immensely distended, while the pulse was 120 and extremely feeble. The temperature continued to rise, and the patient died on

<sup>1</sup> Tait's 'Diseases of the Ovaries,' 4th edit., p. 309.

the afternoon of the same day. At the necropsy I found that the large intestine was very small and had very thick walls, and that there was a piece of small gut adherent to the brim of the pelvis. The small intestine was greatly distended by fluid fæces and gases. The stomach and duodenum were also enormously dilated, and the former had evidently been chronically enlarged. The peritoneum was congested, but there was no visible lymph anywhere, no fluid in Douglas's pouch, and no sticking of peritoneal surfaces to each other, except where the parts had been divided. The explanation of this death seems to be that owing to the contracted condition of the colon and the chronic dilatation of the upper part of the alimentary canal, the muscular power of the gut was placed at a disadvantage. In spite of this the intestine was recovering from the paresis which followed the operation, and the small bowel was able to propel its fluid contents downward into the colon. But when it became necessary to remove the more solid matter which had accumulated in the large gut, the exhausted and embarrassed intestine was unequal to the task. Regurgitation with symptoms of obstruction resulted, and the repeated acts of vomiting produced sufficient stimulation and succussion to move the bowels. When these were well cleared the vomiting ceased; but so much exhaustion had been induced that, before the evacuation of the bowels occurred, the temperature had begun to rise and the patient could not be saved. Had the intestinal peristalsis been aided by an enema, or by a gentle laxative, whenever the vomiting began, or before this, I think that the issue of the case might have been different.

I have made more than one post-mortem examination at the Samaritan Free Hospital, in which the cause of the retention of flatus was not apparent, but in which intestinal distension appeared to be kept up by the pressure of a distended coil on a lower part of the gut. In other cases I have found considerable lengths of small intestine decidedly narrower than the small gut generally. In

one case three yards, from the ileo-cæcal valve upwards, were irregularly contracted, while above this the gut was dilated. There was slight congestion of the peritoneum, but no adhesion, except where parts had been divided, and no fluid in Douglas's pouch. There was no occlusion of the lumen of the gut, for the cæcum was also distended, and flatus had passed from the anus freely during the course of the case. The condition of the intestine, however, must have added greatly to the labour of its muscular wall, and Mr. Meredith informs me that the patient died with all the symptoms of obstruction, except that flatus passed freely from the anus. I have also repeatedly seen—post mortem—the large gut, throughout or in part, much smaller than normal. This I attributed at first to the pressure of tumours, but I have found a similar condition in cases in which the tumours removed had been so small that this explanation could not hold good.

In cases of obstruction of the intestines, whether arising from want of power in the gut or from mechanical causes, the rapidity with which symptoms develop depends mainly on the condition of the intestine and the amount of gas and secretions which are developed in it. As a rule, unless some other complication exist, there is no untoward symptom immediately after the operation. When gases begin to form the semi-paralysed bowel yields at once and painlessly, or with slight colicky pains. The first unfavorable symptom, therefore, may be abdominal distension, occurring about the time when flatus ought to have begun to pass. But if the obstructive condition continue it soon becomes evident that large quantities of watery secretions are being produced by the glands opening into the intestine above the obstructed point. This development of fluid appears to be brought about by a reflex mechanism in the same way as the salivary secretion is often induced before the act of vomiting. Gaseous and fluid matters accumulate in the intestine and distend it till the stomach is no longer able to propel its contents into the duodenum. If food be then given it is imme-

diately rejected with more or less of watery gastric secretions. Sooner or later bilious fluid and the intestinal contents pass upwards into the stomach. Constant vomiting supervenes and an enormous quantity of fluid may be discharged from the mouth, its colour changing as the case progresses from that of the gastric juice to yellow, green, and brown, and finally to a very dark hue, "coffee-ground" or "black" vomit. I have only seen stercoraceous vomiting once in at least five cases of death from obstructed intestine after abdominal section.

As the intestine becomes distended the branches of the mesenteric arteries are subjected to increasing pressure, and this adds greatly to the vascular tension already produced by the inflammatory fever. The heart beats at first more strongly in its endeavours to overcome the resistance thus induced, but the extra work, the depressing effects of the vomiting, and the wasting of elaborate secretions, soon diminish the general and especially the cardiac power, and the pulse becomes small, fast, and feeble.

Thus far the results of retention of flatus and the constant results of the operation are developed side by side, the temperature not being much, if at all, deflected from the course which it usually follows in a case which is doing well. In such a case the temperature reaches its highest point on the second or third evening after the operation and then gradually falls to normal at the end of about a week; so that if the retention of flatus be the only complication, unless the development of gases begin unusually early, the temperature is falling, though perhaps not steadily, when the signs of retention of flatus are becoming prominent. But as exhaustion increases, the inflammation depending on the healing of the parts divided at the operation becomes exacerbated from enfeeblement of resisting power in the tissues, and fever increases. The retention of flatus and the inflammation react harmfully on each other. The bowel, distended to its utmost limits, or to the utmost capacity of the abdominal cavity, becomes quiescent, and all the parts divided

at the operation adhere to adjacent peritoneal surfaces. Finally, inflammation spreads from the wounded parts to the general peritoneum, and as this advances the patient rapidly dies of exhaustion and pyrexia, generally about the fifth day, the symptoms of obstruction having become prominent on the second or third day after operation.

These cases of obstruction are characterised by a remarkable clearness of mental power till within a very short period before death. This is one of the most important signs by which we may distinguish symptoms of obstruction from those of septic origin. Another is the condition of the urine. This secretion is, so far as my experience goes, only partially suppressed a few hours before death in cases of obstruction, and albuminuria does not occur. I have always found a dark colour produced on contact of the urine with strong nitric acid from the presence of excess of indican.<sup>1</sup> The colour becomes more intense as the severity of the symptoms increases, and in cases which recover the reaction becomes gradually less marked as the other symptoms of obstruction pass off.

In these cases the post-mortem appearances in the peritoneum may be normal or those of commencing inflammation. Divided surfaces are usually adherent to adjacent portions of peritoneum, and adjacent coils of intestine may be glued together, but, as a rule, there is no visible lymph formation and no fluid in Douglas's pouch. The intestines are immensely distended by gases and liquids, and a mechanical obstruction of the bowel may or may not be found.

The sequence of events detailed above as following mechanical obstruction of the gut, and the accompanying symptoms, occur in all cases in which death results with retention of flatus after abdominal section. But the symptoms are modified by the nature of the cause of the retention and by the accompanying circumstances.

I now turn to the effects produced by the exudation of blood and serum or of other matters into the peritoneal

<sup>1</sup> 'Parkes on the Urine,' p. 196.

cavity. The exudation of blood and serum in very variable amount is a constant sequence of operations which involve section of the peritoneum. If a case do well, the exuded fluid is quickly absorbed; but if an abdomen be closed when there is much exudation or little absorbing power, or when the tissues are already saturated with serum, as in excessive oedema, the fluid in the peritoneum—even though quite aseptic—may act as a foreign body. And irritating or septic matter invariably does so. Under such circumstances, complete muscular paralysis of the gut is produced, with all the symptoms of obstructed intestine. Distension of the bowel follows, and fever runs high. These signs come together, but the high temperature and rapid pulse which concur with the distension are not due to inflammation but to irritation, and mainly to tension of fluid in a closed sac. That this is so is shown by the facts that the temperature in these cases at once rises when the abdomen begins to distend; but that if a drainage-tube be then passed into the bottom of the pelvis, and the fluid be thereby removed, the temperature immediately falls; and that in exactly similar cases, if Keith's method of drainage be employed from the time of the operation, there will in all probability be little or no fever, for the drainage prevents tension and reduces to a minimum the strain on the absorptive powers of the peritoneum. I have even seen very offensive pus spilt over the peritoneum, and by careful cleansing and the use of the drainage-tube all ill-effects were prevented.

If, on the other hand, the fluid be not removed by drainage, nor by absorption, symptoms of obstruction and of peritonitis rapidly develop. The paralysis of the intestine, the tension and continuous irritation in the peritoneum, with the high temperature and increasing exhaustion, quickly induce general peritonitis, and the patient dies of this disease usually before the fourth day after operation.

In these cases also (unless death be from shock) the



mind usually remains clear, though it may wander from high temperature. The urine may not be suppressed until great exhaustion is induced ; but albuminuria and suppression of urine may occur, for the strain on the kidneys is great both from vascular tension and from the presence of products of inflammation which must be excreted if any attempt at repair be made.

At the necropsy in this class of cases signs of general peritonitis, or at least of commencing general peritonitis, are found, with much fluid in Douglas's pouch ; but masses of lymph and centres of pus formation do not necessarily exist, and when they do, they indicate that some matter more irritating than simple discharges from healthy surfaces has been present in the peritoneum.

If I interpret the signs and symptoms aright, general peritonitis—in the absence of septic influences—is one of the rarest of the primary complications of operations involving division of the peritoneum. Peritonitis must of course occur whenever the peritoneum is divided and does not heal by first intension. But it appears to me that the inflammation which causes fever after abdominal section is mainly in the divided parts outside this membrane, and that the peritonitis which occurs is accurately comparable to inflammation of the skin edges in a superficial wound. When a surgeon speaks of the skin being inflamed in such a case, he means a local inflammation, and when he wishes to imply a more extensive dermatitis, he uses a different word—erysipelas or erythema—or at least a qualifying adjective. So in the case of the peritoneum it is necessary to draw a clear distinction between local traumatic peritonitis and the diffuse inflammation of that membrane which may occur after an abdominal section.

It is of course possible that the necessary exposure and manipulation might cause inflammation of the peritoneum generally ; but the course of the great majority of cases indicates that the discharges into the peritoneal cavity are very rapidly absorbed, and that therefore in

these cases a sufficient surface of peritoneum to effect this purpose remains healthy. When it is necessary to reopen an abdomen a few days after an operation (but of course before the patient is moribund), or when a second operation is required after a prolonged interval, the conditions found also point to the absence of primary general peritonitis. And all I have seen of post-mortem examinations after abdominal section, when considered in connection with the clinical history of the cases, seems to corroborate the view that general peritonitis is rarely produced as a direct result of the operation, but that the peritoneum may be subjected to any necessary treatment without more than a local peritonitis being produced at the divided or severely bruised and adjacent parts. General peritonitis may of course exist when an operation is performed ; and few cases die in consequence of abdominal section (except of shock or hæmorrhage) without evidence of its commencement at least being found at the necropsy. But I have convinced myself, and have endeavoured in this paper to show that general peritonitis after abdominal section is usually, if not always, a secondary effect of the operation, though often it may be the actual cause of death.

Septic peritonitis is a disease arising from a specific influence and communicable from case to case. Mr. Lawson Tait has written a description of this disease following abdominal section. It runs as follows :—“ When from some reason or another the patients begin to do badly, the first indication is an altered expression of the face. I am unable to describe this change of countenance, but I learned to recognise it only too well in the old days, when from the use of the clamp my mortality ran high. Associated with this changed expression is a rapidly increasing abdominal distension, speedily followed by vomiting. At first the vomited matter is simply the fluid the patient has swallowed, but soon it becomes tinged with bile. Later on, should the patient grow worse, the vomited matter becomes entirely bilious, and towards the

end it gets quite black and characterised by those features to which the name of 'coffee-ground' vomiting has been aptly given. In those instances where death followed the use of the clamp, the phenomena always began on the second or third day, the patient dying on the fourth or fifth ; and when once these fatal symptoms had become fairly established, nothing I ever did could arrest them."<sup>1</sup>

These symptoms exactly correspond with those which I have described as due to obstruction, but with the addition of an indescribable change of countenance. The commencement of the symptoms on the second or third day after the operation was due to the origin of the disease. Mr. Tait attributes "the fatal result in such cases to the presence of a minute aperture in the wound at the point where the pedicle was embraced through which the discharges from the ulcerated surface under the clamp penetrated into the abdominal cavity."<sup>2</sup> The date of commencement of the signs of fatal mischief must therefore have corresponded with the date at which the discharges became septic. The similarity in the symptoms to those of obstruction is due to the fact that septic peritonitis at once and completely paralyses the muscular power of the intestine, and so gives rise to what may be called paralytic obstruction in its most marked form. But to complete the account of the symptoms of septic peritonitis, it is necessary to add those due to the effects produced by the poison on the organism generally. The most definite of these are the constant presence of delirium or mental vacuity, the strong tendency to albuminuria, and suppression of urine, the rapid emaciation of the patient, and the marked rise of temperature which precedes death. In fact, in cases of septic peritonitis there are the signs of a triple pathological state, of peritonitis, of blood-poisoning, and of paralytic obstruction of the bowels, the intestinal symptoms being entirely secondary to the paralysis caused by the septic inflammation of the peritoneum.

<sup>1</sup> Tait's 'Diseases of the Ovaries,' 4th edit., p. 308.

<sup>2</sup> Ibid.

The post-mortem appearances are those of intense general peritonitis, usually of a suppurative character.

From a consideration, therefore, of all the circumstances which may attend the operation of opening the abdominal cavity, I conclude that there are two conditions to which retention of flatus may be due in these cases ;—first, mechanical obstruction to its passage down the gut ; second, paralysis of the muscular power of the intestine ; and that this last may arise from over-stimulation during operation, from continued stimulation after operation of the peritoneum covering the gut, or from general septic peritonitis. It is usually impossible to distinguish (except by opening the abdomen) between symptoms due to mechanical obstruction and those due to paralysis of the intestine from over-stimulation during operation ; but the several causes of paralysis and the modes of death therefrom are distinct in their origin, in the course of their symptoms, and in their post-mortem appearances. In any case the intestinal distension is not in itself a disease but merely a result of mischief, though it may in turn become an originating or an aggravating cause of inflammation.

In the management of the intestine in relation to an abdominal section the surgeon must use every endeavour to conserve its power and to decrease its work. To this end it is necessary to thoroughly empty the bowels of all scybalous masses or other accumulations before operation ; and perhaps the best means of doing so is to give a dose of castor-oil with ten grains of Dover's powder. These medicines, or some other form of purgative, should be given three or four days before the operation and repeated two or three days later, an enema being administered on the morning of the operation. If time permit too much attention cannot be paid to getting the bowels into a healthy condition. Masses of fæces may become more or less impacted in the intestine either from costive habits or from the effects of disease ; and the preparatory administration of purgative remedies, or the manipulation

during operation may merely dislodge these scybala from positions in which they have been hitherto harmless. Such scybala are not infrequently passed *per anum* after operation, often with severe pain and alarming fever from disturbance of partially healed tissues. But in the condition of the intestine which follows operation it may be unable to expel these masses, and they may become the cause of complete or partial obstruction and of the death of the patient. I have made one post-mortem examination, in which a scybalous mass was found to have been forced into a constricted portion of the descending colon, where it produced an almost complete obstruction. The constricted part was about four inches long and only admitted one finger. It contained a series of five or six little hard pieces of fæces, which fitted it tightly, and there were large hard masses in the distended bowel above. In the absence of Mr. Thornton I had charge of this patient after the operation, and when she showed symptoms of obstruction I endeavoured to move the bowels by repeated and increasing doses of saline purgatives, but I only succeeded in getting flatus to pass with very temporary relief, and the patient died on the fifth day after operation with all the symptoms of obstruction which I have described. Evidently the constricted condition of the colon which prevented the masses of fæces from coming into the rectum after operation had also caused the accumulation of these masses and had prevented their removal by the preliminary purgation.

During an operation all unnecessary handling and exposure of the peritoneum should be avoided, and every attention should be paid to leaving the abdominal contents as nearly as may be in their natural condition. Every bleeding point should be secured, and Keith's method of drainage should be used whenever a doubt exists as to the power of the peritoneum to absorb the discharges. Of course, too much trouble cannot be taken to prevent contact of the tissues with everything that could possibly convey sepsis to the blood, but it must be remembered

that carbolic acid and Lister's spray producer have a stimulating and chilling effect on the nerves and muscles of the intestine which can only be harmful.

After operation, intestinal rest is obviously desirable on physiological grounds. When the sickness caused by the anæsthetic has ceased, only the most easily digestible food should be given, and if there be any sign of gastric or intestinal irritability, nutrition should be effected entirely by the rectum. Unless under special circumstances contraindicating the use of opium, the best and most rational treatment is to give this drug, or morphia, in sufficient quantity to allay pain. During severe operations there may be much interference with the bowel and mesentery, and laceration of these parts in the separation of adhesions. In such cases, when all bleeding points have been secured, there must of necessity be much temporary disturbance of the supply of blood to the affected and the neighbouring parts of the intestine. But interference with the blood supply causes spasmodic contraction, muscular irritability, or paralysis of the affected parts of the gut in accordance with the condition of the blood in the capillaries, and the amount of other stimulation to which the intestine has been subjected. Under such circumstances, to keep the whole intestine as quiet as possible for a time would seem to be one of the plainest indications. Opium is our best agent for this purpose, and in the absence of complications opiates do not paralyse the bowel, but under their influence flatus passes freely and the abdomen remains soft and flat. The intestinal movements are certainly never abolished by opium in any quantity short of a lethal dose. Peristalsis seems to be of the same nature as the cardiac and respiratory movements in that after a period of rest an imperative necessity arises for its recurrence; but the movements of the intestine are much more irregular than are those of the heart and respiration. Opium prolongs the intervals between the peristaltic waves, and, in an embarrassed condition of the bowel, the prolonged period of rest thus

induced, and the consequent greater accumulation of nervous energy in the automatic motor plexus of the gut, are followed by a stronger, steadier, and more effectual peristaltic contraction. Opium, therefore, after an abdominal section, quiets the action of the gut, secures as much as possible the intestinal rest which is so greatly needed, and tends to prevent those irregular, fruitless, and exhausting efforts which are so undesirable at this time. And if the bowels have been properly emptied beforehand there is no necessity for exertion on their part for some days at least after an operation. Opium may undoubtedly do harm in some cases and in some conditions, but there is abundant evidence of its beneficial action, after abdominal section, if given intelligently while its general and special effects are carefully watched.

If, however, retention of flatus, followed by distension or vomiting, come on in the first few days after an abdominal section, and if, from a careful consideration of all the facts of the case, a simple obstruction in the intestine be diagnosed, its cure may be attempted by rest and sedatives, by stimulating the muscular activity of the intestine, or by reopening the abdomen and giving mechanical relief. But it is well known that the treatment of obstruction of the bowels by sedatives acts slowly, and we have seen that during convalescence from an abdominal section, when once the proper balance is lost between the strength of the bowel and the work to be done by it, when regurgitation in the intestine is taking place, it is only too likely that a train of events leading to a rapidly fatal issue has been started. When symptoms of simple obstruction occur, therefore, so long as flatus passes from the rectum from time to time, and vomiting is not constant, it is possible that by the steady administration of sedatives, and by withholding all food from the stomach, the bowel may so far recover as to be able to remove the obstruction. But if flatus be retained and vomiting or distension come on while sedative treatment is in active operation; if the patient be evidently getting worse, and



especially if the pulse-rate be rising, and symptoms of exhaustion be marked, the time for dependence on the effect of sedatives is past. The progress of the disease towards a fatal issue will now be more rapid than a cure by sedatives can be. At any moment the resisting power in the peritoneum may become so lowered that general peritonitis is inevitable, and it is of no use to have the obstruction removed after such a degree of depression is reached, for the very nature of the cause of the peritonitis in these cases—the progressive debility—renders that disease when thus induced almost certainly fatal. There is therefore not a moment to lose. The only hope is to rapidly clear the bowels, or at least to overcome the obstructive condition before general peritonitis is excited.

This ought to be attempted in the first instance by purgatives, the rectum being of course emptied by enema if it contain any *fæces*. Mr. Lawson Tait says that in his practice vomiting and distension are now extremely rare, and for their treatment he recommends “a small dose of sulphate of magnesia, thirty or forty grains in tepid water, repeated every hour or every other hour until the bowels have moved, or two and a half grains of calomel given every three or four hours until a similar effect is produced.”<sup>1</sup> This treatment is excellent in all cases in which retention of flatus is caused partly by some slight and easily removable mechanical obstruction to the passage of the contents of the bowel, but chiefly by weakness of the gut from exhaustion of the motor nervous centre in its wall. In such cases a timely stimulation of the intestine may produce a most beneficial effect. I have seen several cases in which distension and vomiting came on during convalescence from abdominal section, and in some of which death seemed inevitable under sedative treatment, but which immediately improved and steadily recovered after an action of the bowels had been induced by small doses of saline purgatives. Of course it is im-

<sup>1</sup> Tait's 'Diseases of the Ovaries,' 4th edit., p. 314.



possible to tell what the exact cause of the difficulty in these cases was, but there evidently must have been either a simple exhaustion of the gut or some condition sufficient to seriously obstruct the passage of the weakened intestine, but so insignificant that a very slight stimulation of the peristalsis completely and permanently removed it. Such cases are, I believe, not rare, and the symptoms and ultimate result, though due to weakness of the gut rather than to obstruction, are, if persistent, exactly the same as those of mechanical opposition to the passage of the intestinal contents.

And in these cases the form of purgative is of minor importance, and it matters not how soon after operation it is given,—as a rule, the sooner the better,—provided the treatment be effectual in removing the obstructive condition. This proviso is of the utmost importance; for if for any reason the object of the peristalsis be not accomplished, the extra exertion is necessarily followed by a more decided exhaustion; and the condition of the patient becomes worse than before. There is therefore in these cases good reason for giving a purgative soon, if abdominal distension or vomiting commence. For, if given early, the medicine will act on a comparatively healthy gut and not on one that has lost all tone by excessive stretching; it will act before general peritonitis has set in, and in cases in which this line of treatment fails to give mechanical relief, it will in a short time, by stimulating the secretions intensify the symptoms, and so clinch the diagnosis that the surgeon must at once consider the question of reopening the abdomen in the expectation of finding a definite mechanical obstruction, and being able to relieve it before exhaustion becomes excessive, a course which he might not otherwise feel justified in adopting so soon, but which is now his only hope.

If on the other hand a free action of the bowels can be secured before general peritonitis comes on, and if the obstruction do not return, the difficulty to the circulation is removed, and the powers of nature which have thus

far borne up the patient are strengthened by lessening of work and by nourishment which can again be taken. The good effects thus produced more than counterbalance any ills which may result from disturbance of the healing processes, or from separation of recent adhesions, for any fresh discharges which may be thrown out are speedily absorbed, and the patient may be expected to do well.

But when it is diagnosed that retention of flatus is due not to a mechanical cause but to paralysis of the intestine from irritation of the peritoneum by discharges, then the first thing to be done is to remove the discharges by drainage. It is, however, very much better that, if this be likely to become necessary, a drainage-tube should be inserted at the time of operation. To put one in afterwards is difficult, and must be looked upon as a curative measure of doubtful utility, whereas drainage from the first is a preventive proceeding of tried efficiency. An exit for the discharges having been made, if any mechanical obstruction in the gut be also supposed to exist, if vomiting or distension occur, unless there be marked evidence of peritonitis, a purgative should be given. The unloading of the bowel favours the progress to resolution of the local inflammation in the divided parts, as purgation aids the healing of any wound at a distance from the intestine. And unless general or extensive peritonitis or a doubtful state of vitality in some part of the bowel also exist, no harm will result from the movement of the coils of intestine on each other. This will rather do good by aiding the progress of any free fluid in the peritoneum to the bottom of Douglas's pouch, whence it may be removed by the drainage-tube. Moreover, if adhesions be torn any fresh serum which may be thrown out is in this way at once got rid of. And the fact that adhesions are torn shows them to have formed in such positions that they may have been the cause of retention of flatus, or that they might afterwards have become sources of inconvenience to the bowel, possibly of danger to the patient.

If irritation from within the peritoneal cavity be alone

believed to exist, then the treatment for peritonitis should be employed, and as the stimulation of the intestine is in these cases severe and prolonged the necessity for rest is afterwards great.

When diffuse peritonitis (not septic) is present at the time of operation, or is produced by the operation, or by the irritation of retained discharges afterwards, the treatment should be very different from that of paralytic or obstructive retention of flatus. In the cases now under consideration we have to deal with an inflammation arising from an injury. It is of the nature of a traumatic inflammation, and therefore has a tendency to run a definite course ending in resolution, provided the cause be not persistent, and the patient's strength can be maintained. Tension and irritation must therefore be removed by drainage in these cases also. The most absolute rest possible should be secured to the bowel by the administration of opiates, and this is of course the more necessary if doubt exist as to the vitality of any portion of the gut.

The administration of "a rapidly acting purgative" has been recommended by Mr. Lawson Tait "on the slightest indication of peritonitis after ovariectomy," and Mr. Tait says that under this treatment the peritonitis "disappears."<sup>1</sup> It is not to be denied that a rapidly acting purgative may, possibly, cut short or cure a peritonitis in its initial stage, as it is certain that free purgation does sometimes cure or modify other inflammations. But I have endeavoured in this paper to show that the abnormal conditions to which the intestine is subjected during an abdominal section constitute a sufficient stimulation to exhaust the energy of Auerbach's plexus of nerves; and that in the resulting condition of the bowel may be found the explanation of many symptoms, and of not a few deaths, hitherto unexplained, or erroneously attributed to inflammation and other causes. And I have endeavoured to show how, when this condition gives rise to retention of flatus the administration of a purgative may

<sup>1</sup> 'Brit. Med. Journal,' May 15th, 1886, p. 921.

be followed by the happiest results, preventing general peritonitis by removing its cause. The combination of symptoms which may be induced by this condition of the bowel is nearly similar to that caused by extensive peritonitis; but there are usually sufficient grounds for making a differential diagnosis, and it certainly does not seem to have been satisfactorily proved that in cases of general or extensive peritonitis following abdominal section, the administration of a purgative is advisable. Indeed, this treatment seems exceedingly irrational, for it must give rise to much friction of inflamed serous surfaces, and the risk of rupture, if the bowel be damaged, must be great. But this treatment is also impracticable when severe peritonitis is established. Purgatives act by stimulating the nervous centres in the wall of the gut, or the muscular and secretory tissues directly. These, however, may be already completely paralysed by the peritonitis so that the intestine cannot by any means be stimulated to evacuate its contents.<sup>1</sup> Dr. Keith, of Edinburgh, has told me, and I have his permission to publish the statement, that "in some cases of septic peritonitis, you may give the patient what you will, the bowels won't move." In cases of general peritonitis, therefore, the administration of purgatives can only lead to fretting of the bowel and of the peritoneum, to secretion of more intestinal fluid, to a more profound intestinal paralysis, and to aggravation of symptoms and of danger. Simple general peritonitis and septic peritonitis can both be prevented with much certainty. If, however, this end be not attained the prognosis is extremely grave; but unless some other dangerous complication exist, rest, suitable nourishment, and the removal of irritation by drainage are the rational means of treatment.

Many of the facts and theories which I have brought forward in this paper have been suggested by the post-mortem appearances which I have found at the necropsies of cases of abdominal section at the Samaritan Free

<sup>1</sup> Stirling's translation of Landois' 'Physiology,' 2nd edit., p. 340.

Hospital; but I have to express my thanks to Mr. Knowsley Thornton for permission to publish many observations which I have made while assisting in his practice, and it gives me much pleasure to have the opportunity of saying that to my good fortune in being the colleague and assistant of this master of his craft, I owe my practical knowledge of the art of surgery as specially applicable to the region of the abdomen. I would also add that I am indebted for a great deal of information on this subject to the friendship of the Keiths of Edinburgh, who, when opportunity offered, have given me every facility for seeing their practice, and from whose conversation I have learned much while enjoying their hospitality.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 292.)

**AN UNDESCRIBED METHOD**  
**BY WHICH THE**  
**SUPERJACENT WEIGHT OF THE BODY IS TRANSMITTED**  
**IN**  
**UNITED OR UNUNITED FRACTURE OF**  
**THE NECK OF THE FEMUR**  
**THROUGH AN ACQUIRED ILIO-FEMORAL ARTICULATION ;**  
**AND THE BEARING OF THE PRINCIPLE INVOLVED UPON THE**  
**SURGERY OF THE HIP-JOINT.**

**BY**  
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In 320 subjects which I have examined I found several instances of fracture of the neck of the femur. In all but three union was complete. I was much struck by the fact that in three out of the small number of these fractures of the neck of the femur which I observed, a very considerable proportion of the superjacent weight of the body was transmitted to the femur by means of a very strong and dense process of bone, which projected from the front of the upper end of the femur, and articulated above by a flattened upper extremity with a corres-

ponding quadrilateral depression situated immediately below the anterior inferior spinous process of the ilium, and encroaching to a varying extent upon this process.

The opposing surfaces of bone, as well as the portions of bone immediately adjacent, were intimately united to one another by dense ligamentous tissue. The amount of pressure which was transmitted through this newly-formed articulation varied considerably in the three cases. In that in which the fragments had not united by bone, almost, if not the whole of the superjacent weight was transmitted through the acquired joint. In the two cases in which the fragments had united by bone the amount of weight transmitted by the head of the femur was considerable, in one case equalling, and in the other possibly exceeding that borne by the accessory articulation.

I have not seen this condition described or mentioned in any surgical work, though its relatively frequent occurrence among the number of fractures observed in the 320 subjects dissected would tend to show that such a condition cannot be very uncommon.

That I have not been able to find an instance of it in the museums is, I think, no argument for or against its rarity.

I will now describe and figure the two most typical examples, commencing with the ununited fracture. This specimen, whose anterior aspect is presented in outline by Fig. 1, was taken from the body of a vigorous woman fifty years old. It was quite apparent from the condition of the muscles of the leg and from the density of the osseous system generally, as well as from that of the fractured femur, and from the eburnation of the opposing surfaces of the fragments, that the injury had taken place many years before the death of the individual; that it had not prevented her from continuing her very hard and laborious work up to the last, and that that heavy work was performed without her having suffered any extreme inconvenience from the presence of an ununited fracture of the neck of her right femur.

I mention this last fact, as it is usually supposed that such a condition altogether unfits the sufferer for leading a hard, laborious, and active life. (I am referring here

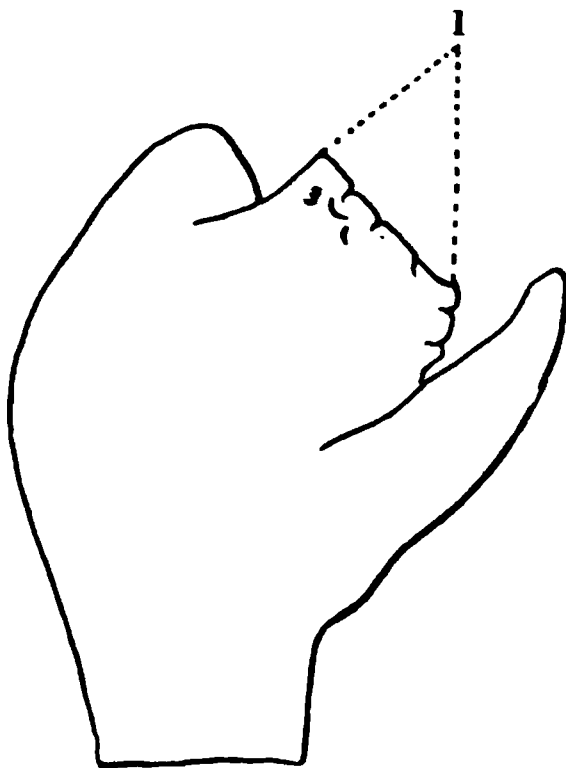


FIG. 1.—The dotted lines indicate the extremity of a projection from the front of the neck. This surface articulated with a depression in and beneath the anterior inferior spinous process of the ileum. The head of the femur is not represented in the diagram.

to ununited fractures of the neck of the femur, in which the opposing fragments, move freely upon one another, and are not connected by intervening ligamentous tissue.)

As I have just stated, the fractured femur as well as the other bones had undergone no degenerative senile change of any sort, but they were, on the contrary, remarkably strong for a woman at any period of life. The compact layer of bone was particularly thick. In such a subject one would certainly not expect to find a well-marked example of intracapsular fracture of the neck of the femur, using the term *intracapsular* in its usual acceptance.

Sir Astley Cooper<sup>1</sup> observed only two cases of intracapsular fracture of the neck of the femur in people below the age of fifty out of a total of 225 instances of this fracture, and, as I have already shown, the liability to fracture of this portion of the neck of the femur in old

<sup>1</sup> 'On the Dislocations and Fractures of Joints,' 1842.



people is due to the fact that the considerable destruction of the cancellous tissue and the thinning of the compact tissue which ensues in feeble old age in both sexes, but more particularly in women, leaves that portion of the neck immediately beneath the head the weakest portion of the bone,<sup>1</sup> and that the supposed alteration in the direction of the neck, which is said to occur in old age and under other conditions, and which has been regarded by many as an important factor in the causation of intra-capsular fracture, is only apparent, and due to an alteration in the form of the head.

In this case the fracture extended through the neck of the femur in a direction from before backwards, and slightly inwards, so that a portion of the neck about three quarters of an inch long was attached to the head in front, while behind the fracture had reached the margin of the articular surface of the head.

The outer surface of the inner fragment was smooth, and was so shaped that its aspect was plane from above downwards, but somewhat concave from before backwards. The facet on the outer fragment was much longer than the corresponding articular surface on the inner, showing that the movements permitted between these portions of the femur were of an upward and downward character. Consequently when the weight of the body was sustained by the injured limb, the head of the femur must have been depressed very considerably below its normal level. This very great vertical displacement of the head was the only attribute common to the three examples of fractured neck which I am describing, and it would therefore seem that it is one of the chief determining factors in the development of the ilio-femoral joint by means of abundant callus.

The stump formed by the neck of the femur was of considerable length, in fact the neck was but slightly shortened by the injury. This fact alone shows that comparatively little strain was thrown upon the opposing surfaces.

<sup>1</sup> "The Causation and Pathology of the so-called Disease, Rheumatoid Arthritis, and of Senile Changes," 'Trans. Path. Soc.,' 1886.

The head of the femur was firmly fixed to the floor of the acetabulum by dense fibrous tissue, so that it required the exercise of very considerable force by means of an elevator to dislodge it. I have already described<sup>1</sup> the manner in which this ankylosis takes place by means of alteration in the character of the opposing lamellæ of articular cartilage, so I will only refer to it here.

An abundant and irregular mass of bone of very great density projected upwards and inwards from the spiral line and from the front of the neck of the femur, and it presented at its upper and inner extremity a slightly concave quadrilateral facet. Its extent and position are indicated by dotted lines and the numeral I in Fig. 1.

This facet fitted into, and was intimately connected by dense fibrous tissue to, a depression of a similar form in and beneath the anterior inferior spine of the ilium. Strong ligamentous bands also united the anterior inferior spine and the adjacent bone to the margin of the bony outgrowth from the femur.

The bone forming the floor of the articular cavity, which had developed in and beneath the anterior inferior spine, was very dense and strong, and it was obvious from a careful examination of the newly-formed joint that very much the greater part of the superjacent weight of the body was transmitted through it, and but a very small amount through the arthrodial joint which had developed in the neck of the femur.

The second specimen, whose anterior aspect is represented in Fig. 2, was obtained from the body of an old woman which was dissected in Guy's Hospital.

The density of her osseous system and the pressure changes it presented showed that she had for long been actively employed and that her occupation had been tolerably laborious.

She appeared to have sustained an impacted fracture of the neck of the left femur, which injury was probably accompanied by much splitting of the great trochanter.

<sup>1</sup> 'Trans. Path. Soc.,' 1886.

The head and neck had been displaced and driven downwards, so that the upper limit of the head lay an inch and an eighth below the upper extremity of the great trochanter, and instead of the neck being directed inwards, upwards, and forwards, it became directed backwards,

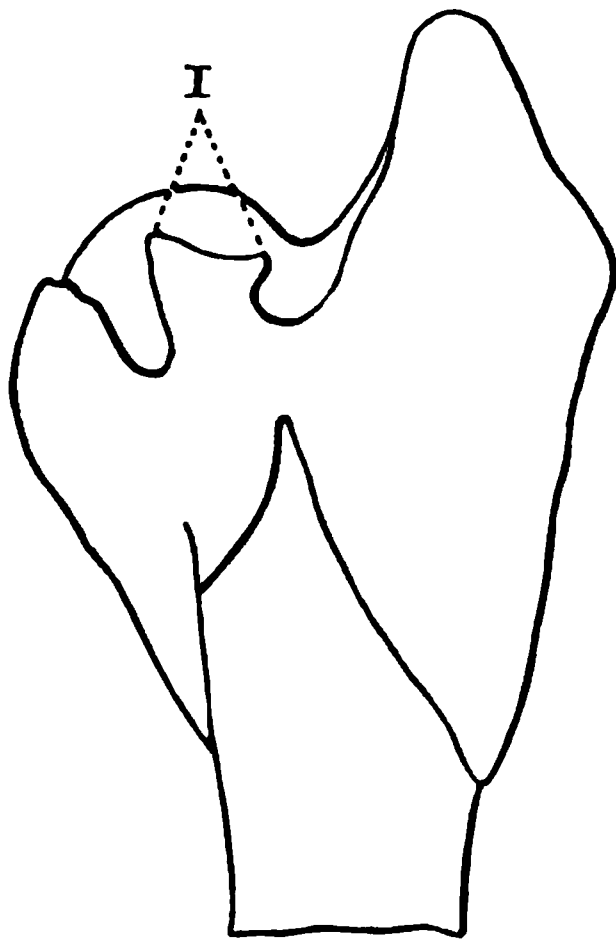


FIG. 2.—The dotted line indicates the surface which articulated with the depression beneath the anterior inferior spinous process.

downwards, and inwards. It is obvious that such an alteration in the direction of the neck would interfere very considerably with the security of the hip-joint and with its capacity for transmitting pressure, especially in certain attitudes.

A somewhat irregular but very dense process of bone projected upwards and slightly inwards from the anterior intertrochanteric line, and ended in two prominences. The outer one terminated in a broad transversely oval facet, whose extent and position are indicated by the dotted lines and the numeral I in Fig. 2. This surface articulated with a correspondingly excavated facet beneath the anterior inferior spine of the ilium, and the opposing bony surfaces were covered by soft fibrous tissue, their margins being firmly connected by a dense

capsule of ligamentous tissue. The internal prominence was pointed, and was connected by ligament to the innominate bone.

It was apparent from the examination of this specimen that a very large proportion of the weight of the body was transmitted through the ilio-femoral articulation, though the proportion was not so great as in the previous instance.

As I said before, I believe that the unusual very considerable downward displacement of the head of the femur, which was present in this as in the other two cases, was the factor which determined the formation of an ilio-femoral articulation in front of the normal hip-joint.

I have described in the 'Guy's Hospital Reports' for 1886 the skeletal changes which are consequent upon a shortening of one limb and upon an excessive compensation of that shortening. In the three cases I have described, the deformity of the trunk which followed on the injury was very slight as compared with that usually present in hardworking people.

In the majority of cases of fracture of the neck of the femur the amount of shortening is usually less than in the three cases which form the subject of this paper, so that in the vast majority of cases the displaced head and neck are still able to perform their normal functions, after they have in many instances been supported by the development of a dense mass of bone below the displaced neck at its junction with the shaft, so that there does not then exist the same necessity for the presence of any accessory joint.

I would now direct particularly the attention of the reader to the immense mechanical advantages which the ilio-femoral articulation affords in cases of fracture of the neck of the femur in which there is much downward displacement of the head of that bone, and these advantages are especially obvious when the fragments move freely upon one another. I would also ask him to consider the

manner in which this newly-developed articulation serves to diminish as much as possible the shortening of the limb both apparently and practically, to render it very much more useful than it would be without the presence of such a powerful joint, and to reduce to a very considerable extent the deformity of the trunk, and particularly the extreme lordosis and unsteady gait which would otherwise ensue on the injury. I believe that the application of the knowledge of this mechanism will enable us to make very considerable strides in the operative surgery of the hip-joint.

Let us take, for example, the operation of excision of the hip-joint. We are all only too familiar with the very unsatisfactory result of the most successful operation and with the comparatively useless condition of the excised limb. Apart from the shortening, what is the chief reason of the practically complete failure of this operation?

I think the cause is perfectly obvious. The divided upper extremity of the femur is left in apposition with the outer aspect of the ilium, to make for itself a most insecure and unsatisfactory connection with that bone in a position in or behind the original vertical plane of the acetabulum. A secure or firm joint is never or hardly ever formed, but there develops instead a new joint which is arthrodial or amphiarthrodial in character, and whose flat articulating surfaces are placed almost vertically. This new articulation, on the security of which the usefulness of the limb depends, allows of a gliding movement between the bones, the upper extremity of the femur rising vertically to a variable extent when the weight of the body is thrown upon the excised limb.

During locomotion the rotation of the pelvis around its antero-posterior and transverse axes is much in excess of that normally present, so that this joint is made to support a strain which is considerably greater than that borne by its healthy fellow. This strain is also thrown upon it at a great mechanical advantage, and in a ver-

tical plane which is anterior to that in which the acquired joint lies, so that everything combines to diminish its security and usefulness. The only obstacle to the upward displacement of the femur is a dense ligamentous capsule or strong intervening interosseous ligament.

It is quite obvious, from a careful examination of the mechanism of this ilio-femoral articulation which developed in the case of ununited fracture of the femoral neck, that if in the operation of excision of the hip-joint the upper extremity of the femur were displaced upwards, forwards, and inwards, and were attached or fixed in position to the anterior inferior spinous process and to the bone immediately beneath it, we would have formed an ilio-femoral articulation which would differ in no way from, and would serve its purpose quite as well, as did that which I described in the ununited fracture in the neck of the femur. That some difficulty might be experienced in uniting the upper extremity of the femur to the innominate bone and in retaining it in position is very possible. I am unfortunately unable to speak from a practical experience upon this point, as I have not recently had an opportunity of applying my suggestion, yet I have not the slightest doubt, with our present facilities of surgical treatment, these difficulties would be readily and effectually met. It is apparent that the mechanical advantages which would be gained by the formation of such an ilio-femoral joint in cases of excision are simply enormous. Instead of the usually very feeble joint which, when unsupported, cannot, except in very rare instances, support the weight of the body, we would have an articulation which would sustain nearly as much strain as the original, which would be accompanied by much less deformity of the trunk both in standing and in walking; and the deficiency in the length of the leg could be readily compensated without increasing the upward displacement of the femur and diminishing still further the usefulness of the joint in connection with that bone.

Other possible applications of this principle suggest

themselves, but the one I have described serves to illustrate it sufficiently well for my purpose.

I have illustrated in the following diagrams the appearances usually presented by united and the various forms of ununited fractures, as the outline indicates pretty plainly the mechanism of each.

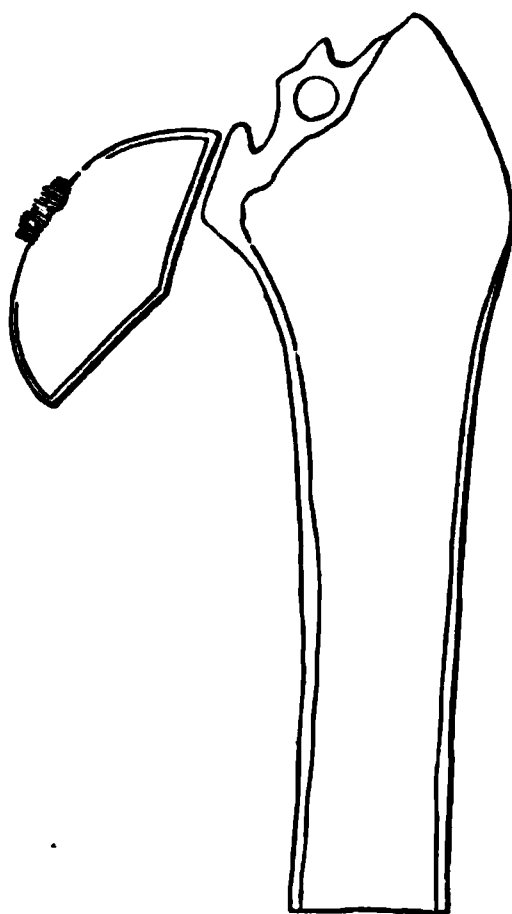


FIG. 3.—Ununited fracture of the neck of the femur. The upper extremity of the femur was connected to the innominate bone by a dense fibrous capsule, but the head was not connected to the femur in any way.

Fig. 3 represents a vertical transverse section through an ununited fracture of the femoral neck. The neck had been completely removed by the friction of the fragments upon one another. The head was fixed in the acetabular cavity by the shortened ligamentum teres, and its articulating external aspect was flush with the margin of that cavity. The upper extremity of the femur was rounded and was connected to the innominate bone by a very dense ligamentous capsule, whose upper and back part was very much thicker than the remainder. The conditions present are exactly analogous with those seen in many cases of excision of the hip-joint, the upper and back part of the capsule being the only mechanism by which upward dis-

placement of the femur is obviated. The very unsatisfactory character of such an arrangement is only too apparent.

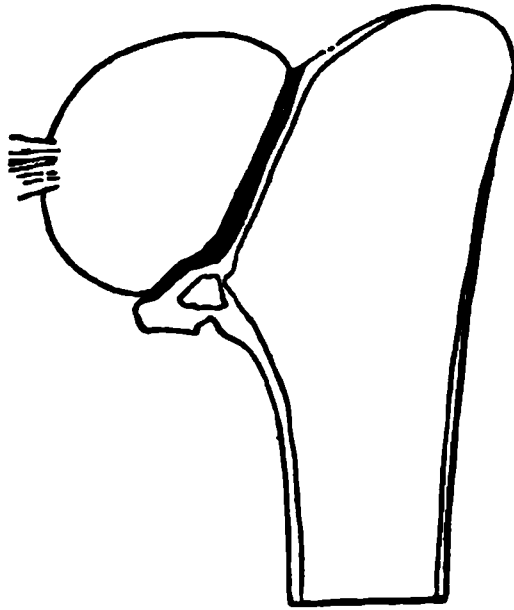


FIG. 4.—Vertical transverse section of an ununited fracture of neck of femur. The upper extremity of the femur and the outer surface of the head were united by a layer of dense ligamentous tissue. A process of bone projects inwards beneath the head and prevents its depression.

Fig. 4 represents a similar transverse section of an ununited fracture of the neck of the femur in which the fragments were connected by a layer of dense ligamentous tissue, and the downward displacement of the inner fragment was obviated by the presence of a supporting shelf of bone which projected inward from the lower fragment.

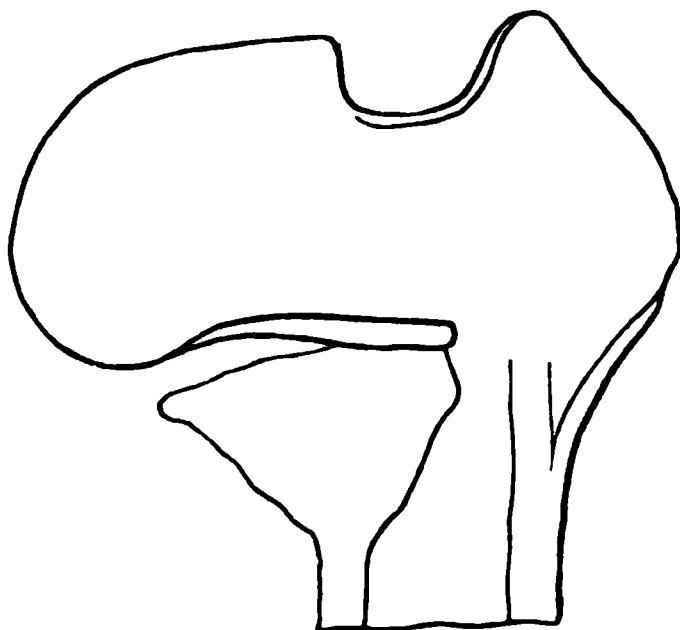


FIG. 5.—Transverse vertical section through a united fracture of the neck of the femur. As in the other diagrams the double outline indicates the thickness of the compact layer.



In Fig. 5, which represents a united fracture of the neck, the displaced head and neck are sustained by a mass of callus similar in position and function to that present in Fig. 4.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 302).

A CASE  
OF  
EXCISION OF A TUBERCULAR MOVABLE  
KIDNEY.

BY  
WARRINGTON HAWARD,  
SURGEON TO ST. GEORGE'S HOSPITAL.

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Received April 12th—Read November 22nd, 1887.

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ANNIE M—, æt. 16, was admitted into St. George's Hospital under my care on November 20th, 1886. She came of a healthy family, and excepting that she had been liable to sick headaches, had been fairly healthy till July, 1885. About that time she began to be troubled with increased frequency of micturition, and the urine became thick and offensive. During the ensuing twelve months the irritability of the bladder increased; and to this was added pain in the bladder and urethra, both before, during, and after micturition; subsequently she also began to suffer pain in the right loin. The urine occasionally contained a little blood.

She did not lose flesh, had no cough, and ascribed all her trouble to the bladder.

On admission; she was a pale but well-nourished girl,

showing no marks of scrofula. She was obliged to pass water very frequently, sometimes every half-hour, or even at less intervals, and more often when up than when recumbent.

The urine produced a burning sensation when passed, but she always felt more comfortable for a time after the bladder was emptied. The amount of urine passed averaged about 26 ounces in twenty-four hours. The sp. gr. = 1014; reaction neutral or faintly acid. It deposited on standing about a tenth of its bulk of pus: under the microscope it was seen to contain a few blood-globules and crystals of triple phosphate, and a large quantity of pus.

Examination of the abdomen revealed a somewhat oval-shaped tumour about six inches long and four inches broad, which usually occupied the right lumbar region, but which could be easily made to move either up under the ribs, or down into the right inguinal region, or across to the umbilical region so that its edge projected a little to the left of the middle line. The tumour felt in some parts elastic, in other parts firm and even hard; its outline was lobulated, its inner border rounded; and it could be easily moved between the fingers of the left hand posteriorly, and those of the right hand in front of the loin. Its general shape, character, and position corresponded with those of an enlarged and movable kidney. Pressure upon the tumour produced slight pain and a feeling of nausea.

There was no evidence of any other abdominal or thoracic disease. The bladder was sounded with negative results.

Temperature and pulse were normal.

The diagnosis was suppuration, probably of a scrofulous character, affecting a movable kidney. But as the girl showed no other signs of scrofula, it seemed doubtful how far the enlargement and inflammation of the kidney might depend upon its mobility, and the consequent disturbance of the circulation and impediment to the outflow of urine

which might thus occur. A belt was therefore applied with a suitable pad for keeping the kidney as much as possible in its natural position. Large quantities of distilled water were also administered. The patient was moreover kept at rest in bed.

For a short time the rest, and the dilution of the urine seemed to give her some relief, but the pressure of the belt caused her a good deal of local discomfort. Towards the end of December she again became worse, the irritability of the bladder and pain causing her constant distress. It was also observed that whenever the kidney was subjected to manual examination a rigor immediately followed, and the temperature rose several degrees ; for example, to  $102.6^{\circ}$ , to  $103^{\circ}$ , to  $104.4^{\circ}$ , on different occasions, subsiding again to the normal after the lapse of a few hours.

During the next month henbane, opium, pareira, and other drugs were administered to combat the irritation of the bladder, but none of these remedies did the least good. The pain and frequency of micturition steadily increased and the aspect of the patient became wearied and anxious.

It seemed, therefore, that the best chance of relief would be given by the removal of the diseased kidney ; and in consultation with my colleagues this was agreed upon.

The operation was performed at 9.15 a.m. on February 17th, 1887, with the assistance of Mr. Dent ; careful antiseptic precautions were observed, but no spray was used. The bowels had been previously cleared by castor-oil and enemata, and the patient was directed to pass water immediately before the operation.

Ether was administered, and an incision was made two inches to the right of the middle line, extending from about two and a half inches above to the same distance below the level of the umbilicus. This was intended to be in the semilunar line, but at the upper part a few fibres of the rectus were cut. The abdomen having been opened the kidney at once presented covered with its layer of

peritoneum. This was divided and the finger was then swept round the kidney so as to separate it from the surrounding tissue, to which it but loosely adhered. The outer border of the kidney projected forwards, so that the pedicle was behind the organ; by tilting it a little outwards, however, the renal artery could be felt pulsating, and the dilated commencement of the ureter could be seen. A double silk ligature was passed between the ureter and the vessels and tied on either side; the kidney was then brought out of the wound and cut away. As the ureter was divided great care was taken to prevent any of the fluid in the pelvis of the kidney passing into the wound, which was protected by sponges packed closely round the kidney.

Scarcely any blood was lost during the operation, the only vessels requiring ligature being two or three small arteries in the abdominal wall. But when the ligature was tightened round the pedicle the pulse became imperceptible for several beats, and then very rapid and feeble. It soon, however, recovered. Before replacing the pedicle within the abdomen the cut ureter was examined, and as it was found to be thickened and lined with a layer of secreting granulation tissue, it was separated from the rest of the pedicle and attached to the surface of the upper angle of the wound. The rest of the wound was then accurately closed with silk sutures, the end of the ureter dusted with iodoform, a dry pad of salicylic wool in carbolised gauze applied, and a many-tailed flannel bandage firmly adjusted over the whole abdomen.

Nothing was seen during the operation of the other contents of the abdomen, excepting just the border of the right lobe of the liver and the edge of the ascending colon.

The kidney removed is of lobulated form, and is hollowed out into a number of large rounded cavities the walls of which, bulging towards its surface, are in some places not more than a quarter of an inch in thickness. It was seen during the operation that the walls of these

cysts were so thin and soft that a very little pressure or handling would have ruptured them.

On laying the organ open its interior was seen to be filled with a quantity of creamy fluid and caseous material, and to be lined with a vascular layer of granulation tissue. Very little of the secreting structure remains, but at the lower and outer part there is a small portion of cortex to be seen, which is studded with numerous small round caseous deposits, varying in size from a pin's head to a pea. The weight of the kidney when emptied of its fluid contents was eight ounces.

The pelvis and commencement of the ureter is dilated and thickened.

Dr. Délépine has been kind enough to make a microscopical examination of some of the remaining cortex, and reports that it exhibits "a condition of progressing interstitial nephritis, in the midst of which are numerous small growths showing the usual characters of tubercle; several of them contain giant-cells, and others are beginning to undergo cheesy degeneration."

For the first twelve hours after the operation the patient was fairly comfortable. At 3 p.m. she passed involuntarily a few ounces of urine, which was absorbed by an antiseptic pad placed ready in case of need. She was thirsty, and was allowed to wash out her mouth with tepid water tinged with Condy's fluid; and as she had some pain in the loin, an opiate enema was given.

At 11 p.m. she had passed water twice voluntarily and was comfortable and inclined to sleep. Pulse 118, temp. 99°.

During the morning of the next day (Feb. 18th) she vomited three times a little blood. She was therefore given ice to suck.

The vomiting recurred in the evening. It seemed as if a little blood oozed into the stomach and was then vomited. There was very little effort or straining, and the vomiting seemed more in consequence of the presence of blood in the stomach than of a kind to provoke hæmorrhage.

Ergotine was therefore subcutaneously injected. The vomiting of small quantities of blood still, however, continuing, small draughts of hot water were given, and subsequently hydrocyanic acid, but without effect.

Enemata were given every four hours, each containing two ounces of beef-tea thickened with starch, half an ounce of brandy, and twenty drops of laudanum. During the night the temperature rose to  $100\cdot6^{\circ}$ . The vomiting continued, and she sank on the morning of Feb. 19th, forty-four hours after the operation.

Between the time of operation and death urine was passed at intervals of three or four hours, sometimes voluntarily, at others involuntarily, into antiseptic pads. That which was collected was coloured and turbid as if from the presence of lithates, but did not look as if it contained blood. It was put aside for examination and measurement, but by an unfortunate mistake was thrown away. As far as can be ascertained, the whole quantity probably amounted to eleven or twelve ounces. The last was passed involuntarily about half an hour before death.

The post-mortem examination was made thirty-three hours after death. The body was well nourished. The edges of the abdominal wound were everywhere firmly adherent. There was no peritonitis. The ligature was securely embedded in the pedicle, at a spot about two inches distant from the aorta.

With the exception of a few streaks of blood in the perinephral cellular tissue all the neighbouring parts were quite natural.

The left kidney was quite healthy, and showed nothing unusual excepting congestion of its blood-vessels, which was evidently quite recent. It weighed four ounces. The ureter was natural. The right ureter was natural with the exception of its upper inch and a half which was dilated and thickened.

There were a few patches of congestion on the mucous surface of the bladder near the entrance of the ureters; the organ was otherwise healthy, as was also the urethra.

There were several areas of congestion, about the size of a florin, on the mucous surface of the stomach. The organ was in other respects natural. With the exception of some firm adhesions of the left pleura, all the other thoracic and abdominal organs were quite healthy.

For the proper estimation of the value of a new operation, it is necessary that all the cases in which it is practised should be published; and as the operation of excising the kidney is a comparatively recent one, I have thought it desirable to publish this case, especially as the operation was unsuccessful, and as it seemed to me therefore, that some instruction might be obtained by others as well as myself by its being submitted to the criticism of this Society.

One feature of interest in the case is the fact that the disease was limited to this one organ, and that even the ureter was affected only at its commencement, although the affection of the kidney had existed probably for sixteen months or more. The rest of the genito-urinary tract was unaffected by any morbid change, with the exception of the small area of congestion in the bladder, and there was no sign of tubercle elsewhere in the body. But the irritation of the bladder by the tuberculous pus was very great and was increasing; and seeing that there is good reason to believe such pus to be locally infective, this furnished one strong argument in favour of removal of the kidney.

There was, of course, the other alternative of opening and draining the kidney in the loin, but the great mobility of the organ, and the probability that its secreting tissue was extensively destroyed, seemed to point to its removal as the better course.

Moreover, the observation of other cases in which the kidney had been drained through an opening in the loin, showed that though this is eminently satisfactory in calculous and some other forms of pyelitis, that in scrofulous pyelitis, though the relief may be very great, yet it is difficult to prevent some of the irritating pus still finding



its way down the ureter and keeping up irritation of the bladder.

The abdominal incision was preferred to the lumbar chiefly because the size of the kidney would have rendered it difficult or impossible to have extracted it entire through the interosseous space in the loin, and because, also, of its displacement towards the front of the abdomen.

It would probably have been better, instead of attaching the ureter to the wound, to have turned inwards its coats, sewn up its end, and returned it with the rest of the pedicle.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 306.)

**A CONTRIBUTION**  
**TO THE THEORY OF THE**  
**NERVOUS ORIGIN OF RHEUMATOID**  
**ARTHRITIS.**

**BY**  
**ARCHIBALD E. GARROD, M.A., M.D.**

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AMONGST those whose attention has been directed to diseases of the joints there is an impression, which is steadily gaining ground, that the disease which we know as rheumatoid arthritis, osteo-arthritis, or arthritis deformans, is in an especial manner associated with some lesion of the central nervous system.

This view, first advanced by the elder Remak and by Benedikt, was advocated by Senator in his masterly article in 'Ziemssen's Handbuch,' and has been placed upon a firmer basis by the researches of Dr. Ord, Sir Dyce Duckworth, Mr. McArdle, Dr. Latham, and Dr. Weber of New York.

The arguments which have been put forward in support of this theory may be divided into four classes :

First : Those based upon the clinical characters of the disease ; its causation ; the distribution of the articular lesions, and the absence of visceral complications.

Secondly : Those based upon various nervous phenomena associated with this disease, such as trophic changes in the muscles, bones and skin, the exaggeration of tendon-reflexes, painful muscular spasms, and the occasional occurrence of migraine and palpitation of the heart.

Thirdly : Arguments based upon the results of treatment, especially those obtained by some observers by the use of electricity.

Fourthly : Arguments based upon the similarity of the morbid changes observed in rheumatoid arthritis, to those resulting from joint affections more certainly of nervous origin.

Whilst reading the literature of rheumatoid arthritis it occurred to me that it would be interesting to examine some of these arguments in the light of a large number of observations, and I have collected a series of 500 cases, for the notes of which I am indebted to my father, Sir Alfred Garrod, upon which, as well as upon cases which have come under my own observation, are based the results which I propose to lay before the Society.

This series includes all the varieties of rheumatoid arthritis, from the non-articular disease of elderly people to the most extensive and general form of the complaint.

The points which I hope to bring out are the following :

First : That the causes of rheumatoid arthritis are such as might be expected to act upon the central nervous system.

Secondly : That the distribution of the joint lesions is such as would be likely to result from nerve lesions.

Thirdly : That the distribution of the lesions is similar to that of certain arthropathies of spinal origin.

The following case, which has recently been under my care, illustrates so many of the points to be discussed that I venture to give the notes in some detail.

A lady, æt. 46, with no hereditary tendency to joint disease, had always had good health until four years ago. At that time she spent six hours in a state of intense anxiety and excitement, and when her anxiety was relieved,

was seized with a violent hysterical attack, although she had never before shown any symptoms of hysteria. She was ill for some days afterwards, and four months later the joint trouble commenced. The catamenia, which had up to that time been perfectly regular, ceased at the same time. The joints of the feet were first affected, and almost simultaneously stiffness of the jaw was noticed; then the ankles, knees, hands, wrists, elbows, shoulders, and hips were successively attacked, the disease working up the limbs from the periphery.

The pain was at first confined to the affected joints, but afterwards was felt along the bones, and lately there have been attacks of intense spasmodic pain in the muscles around the right hip.

The deformity is extreme, and all the joints are limited in their range of movement. The hands are much distorted although the joints are hardly appreciably enlarged; the fingers are deflected to the ulnar side, and in the right hand the first phalanges are flexed upon the metacarpals, the second phalanges are extended upon the first, and the third are somewhat flexed upon the second, whilst in the left hand there is flexion of nearly all the joints. A sharp osteophyte upon the second knuckle of the right hand threatens to pierce the skin. There is much distortion of the toes and slight œdema of the legs.

The muscular wasting is most marked in the muscles of the hands and forearms. The finger-nails are dull, fibrous, and brittle, and the skin of the fingers is glossy and pink, but sensation is not impaired. The glossiness of the skin and the muscular wasting were noticed almost immediately after the joints of the hands became affected. There is no sign of any visceral disease; the heart's action is weak but there is no murmur. All the joints of the extremities being involved, I am unable to give any account of the tendon-jerks. The electrical reactions show nothing abnormal.

In the study of any form of joint disease the question

of the influence of heredity is one of extreme interest, but in the case of rheumatoid arthritis it is rendered very difficult by the vagueness of the notion entertained by the public on the subject of "rheumatic gout."

Sir Dyce Duckworth and Sir Alfred Garrod have expressed the opinion that the daughters of gouty parents are frequently attacked by rheumatoid arthritis, and the view of the former author, that the members of certain families are especially liable to fall victims of joint disease of whatever variety, does not appear to me to be in any way opposed to the nervous origin of rheumatoid arthritis, since we may readily suppose that the joints of those who inherit the arthritic diathesis would act as points of least resistance, and would become the seats of an arthritic process, as the result of disturbances of the nervous system insufficient to produce such changes in persons not so disposed.

For reasons above mentioned the following table, which contains the results obtained from the study of 500 cases, cannot claim more than an approximation to accuracy :

Family history of gout . . . . .	64
„ „ doubtful gout . . . . .	10
„ „ rheumatism . . . . .	48
„ „ rheumatoid arthritis . . . . .	11
„ „ hand-joint affection . . . . .	14
„ „ joint affection . . . . .	24
„ „ rheumatic gout . . . . .	1
„ „ deformed joints . . . . .	1
„ „ crippling by rheumatism . . . . .	11
„ „ sciatica . . . . .	2
„ „ gout and rheumatism . . . . .	7
„ „ gout and hand affection . . . . .	5
„ „ gout and crippling . . . . .	2
„ „ gout and joint affection . . . . .	1
„ „ gout and deformed joints . . . . .	6
„ „ gout, rheumatism, and rheumatoid arthritis . . . . .	1
„ „ rheumatism and joint affection . . . . .	4

Family history of rheumatism and sciatica.	.	1
„ „ rheumatism and crippling	.	1
„ „ rheumatism and rheumatoid		
arthriti s . . . . .	.	1
„ „ rheumatism and hand affection		1
		<hr/>
		216

It will be seen that in 216 cases, or rather less than half, there was a family history of arthritic affections; of these gout holds the first place, being met with in nearly one fifth of the cases. Rheumatism occurs sixty-four times, but some of the conditions so named may very probably have been in reality rheumatoid arthritis. Of the remaining conditions, such as crippling by rheumatism, hand affection, deformed joints, &c., which may be grouped together as probably rheumatoid arthritis, there are eighty-four examples, placing this disease second to gout.

After allowance has been made for the fact that in any 500 people such family histories would frequently be met with, it still seems to me that it must be allowed that an arthritic tendency does play an important part in the causation of rheumatoid arthritis, and this conclusion is borne out by individual instances.

I have at present under observation a poor woman who presents a typical example of rheumatoid arthritis. Of her grandparents she knows nothing; her father and mother both had enlarged joints. She herself is one of fifteen children, six of whom grew up, and her three brothers and two sisters suffered from enlarged joints as she does.

In another case, that of a young woman, æt. 24, who is entirely crippled by the disease, I obtained the following history. Her father was rheumatic, her mother had a rheumatic affection of the knees, one brother died of rheumatic fever, and one sister had a rheumatic affection of the ankles.

I have not found that those patients in whom the dis-

ease begins early are more likely to have a family history of joint disease than those in which the first symptoms appear later in life.

The fact that only a part of the patients who have an arthritic family history inherit the actual disease from which they suffer, seems to me to be strongly in favour of the existence of a general arthritic diathesis.

To pass on to more immediate causes, no one who has read what Dr. Ord has written upon the subject can doubt that uterine disorders are among the most potent causes of rheumatoid arthritis, and their importance has been recognised by many other authors.

Of 176 cases in which the state of the menstrual function is noted, it was regularly performed in 105, and was irregular in period or in quantity in 71.

Of the women 267 were married and 144 single.

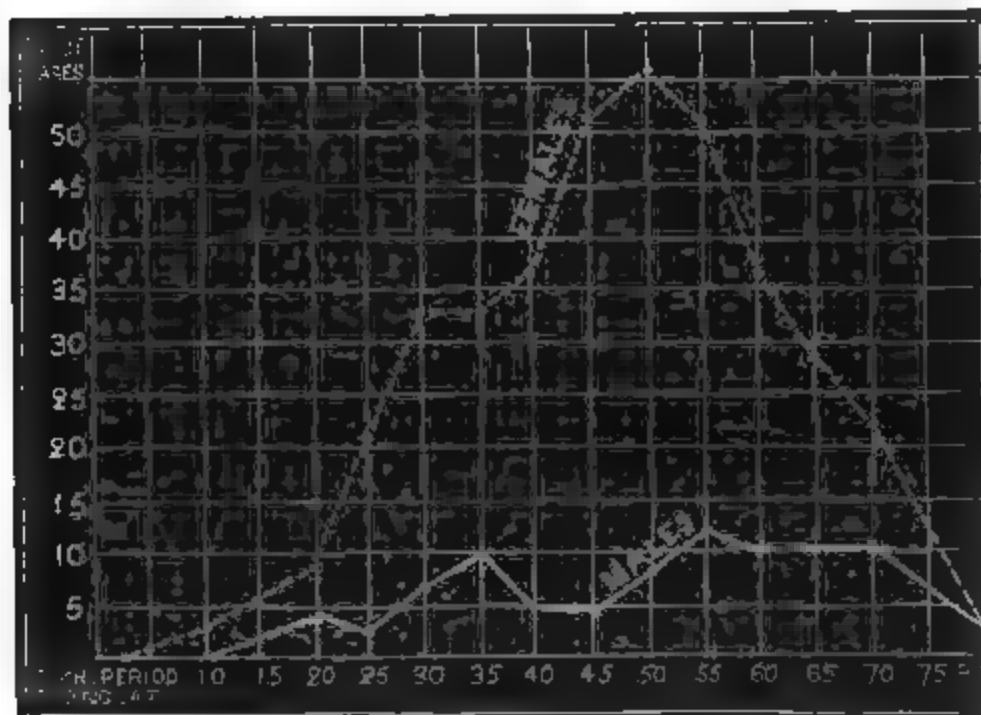
In 132 cases the extent of the family was noted, and of these 92 had less than six children, and 40 more. Two had thirteen, one fifteen, and one seventeen children and three miscarriages.

In five cases the disease was stated to have begun after a confinement, in three after a miscarriage, and in three during pregnancy; in two others the disease was worse after confinement, and in one during pregnancy, whilst one patient stated that she suffered less during pregnancy. Five gave a history of uterine or ovarian tumours.

The point which I am able to bring out most clearly is the influence of the menopause on the development of the disease, a fact to which Haygarth first drew attention. Eighteen patients dated its onset from a period within two years before the menopause, and forty-one from the two following years, and in five cases the two events were simultaneous.

The influence is, however, best illustrated by the following table and curve, which show the ages at the commencement of the disease of the male and female patients respectively.

Age at commencement.		Women.		Men.
0—9	...	8	...	0
10—19	...	18	...	4
20—24	...	21	...	8
25—29	...	33	...	7
30—34	...	33	...	10
35—39	...	37	...	5
40—44	...	51	...	6
45—49	...	57	...	8
50—54	...	51	...	12
55—59	...	37	...	10
60—64	...	29	...	10
65—69	...	23	...	10
70—80	...	2	...	3
80—90	...	1	...	0
Doubtful	...	15	...	3



Curve illustrating the above table.

It will be seen that in the female cases there is an unbroken rise till the five-year period 45—49 is reached, and afterwards an unbroken fall. In the male cases, on the other hand, there is no such regularity, the numbers tending to increase with age, with a rise between 20 and 30, a period mentioned by both Charcot and Lebert as one at which the disease is likely to begin.



The table shows moreover the far greater liability of women to the disease, the numbers being 411 women to 89 men.

The influence of worry and care in the causation of rheumatoid arthritis has been insisted upon by many writers, and it is adduced by Senator as an argument for the nervous origin of the disease. Such a history is seldom volunteered by the patients, and is often only to be elicited by careful inquiry.

I find, among the 500 cases, 34 in which such causes are mentioned, including bank failures, sick nursing and the like.

For the following instance I am also indebted to my father, but it is not one of those already mentioned.

A lady, aged between fifty and sixty, who had always enjoyed good health, lost her only daughter after eight or nine months' illness. The mother's grief was intense, and almost immediately after the daughter's death she was attacked by rheumatoid arthritis of both knees and the left elbow. In the course of a year or two her health was restored, but the affected joints were left perfectly stiff.

The influence of violent mental shock is most important from a theoretical point of view, and the cases related by Koht<sup>1</sup> have been repeatedly quoted. The patients were two men aged thirty-eight and twenty-six respectively, who developed well-marked rheumatoid arthritis soon after receiving intense shocks from the bursting of shells, one during the bombardment of Strasburg, the other during the Paris Revolution in 1848. Leyden<sup>2</sup> also states that in Strasburg many cases of this disease date their onset from shocks received during the siege of 1870.

It is at least possible that the illness of the lady above mentioned, in whom the disease began after a short period of intense anxiety, may be attributed to this cause.

<sup>1</sup> 'Berliner klin. Wochenschrift,' 1873, p. 304.

<sup>2</sup> 'Klinik der Rückenmarkskrankheiten,' p. 159.

In the series of cases there are two in which the commencement of the disease was preceded by a severe mental shock, the nature of which is not, however, specified.

The influence of accidents and injuries is most marked in the local variety of the disease, for most writers agree that chronic rheumatoid arthritis of the hip-joint usually follows an injury slight or severe.

In this connection the following case, for the notes of which I am indebted to Mr. Thomas Smith, is of interest.

The patient was thrown from his horse, which fell upon him, bruising his leg severely, and straining the hip. The accident occurred in South Africa. His medical attendant did not think seriously of the injury at the time. A month after the injury he sailed for England, and on his arrival was seen by Mr. Smith, who found much bruising of the adductor and other muscles but no sign of hip disease.

He recovered sufficiently completely to allow of his going out shooting nine months after the injury, and three months after that he hunted. Although walking a good deal he had to rest at times on account of his leg.

Two years and a quarter after the injury he began to suffer severely from the hip, but was able to ride for some time after this. The hip affection has steadily progressed, and he now presents a perfect example of chronic rheumatoid arthritis of the hip-joint.

The researches of Dr. Charcot, which show that injury to a joint is followed by dystrophy of the neighbouring structures, together with those of Berger, on the arthritis which follows fractures of bones, have been adduced as pointing to a close relation between the condition of the joints and that of the surrounding structures, so that injury to one may be followed by a reflex affection of the other. If this be so, it seems to me that we have here, at least a plausible explanation of the frequent occurrence of the local variety of rheumatoid arthritis after injuries of the neighbouring parts.

Nor are cases wanting in which the onset of general rheumatoid arthritis is attributed to an injury.

For example, a woman, æt. 30, whose right foot and hand and neck were affected, dated the commencement of the disease from an accident. In a man, æt. 45, whose hands, elbows, shoulders, feet, ankles, knees and neck were all affected, the disease began after a fall which caused a scalp wound and shock. In another case the disease invaded the fingers of the right hand after an injury to the fourth finger.

A similar case is recorded by Mr. McArdle, of Dublin.<sup>1</sup>

Cold and damp have long been acknowledged as causes, but the cases in which I find this mentioned are only nine in number. One patient had lived in a damp part of Ireland; one had got wet and cold during a menstrual period; two attributed their troubles to sleeping in damp beds; two lived in damp houses before the disease began; two attributed the disease to exposure to cold, and one to drinking cold water when hot.

A case, quoted by Dr. Ord,<sup>2</sup> of a lady who sat for some hours, in evening dress, with her back near the wall of a damp marquee, also illustrates this point.

Dr. Charcot, in his thesis of 1853, laid great stress upon this cause, but he considered that the action of cold and damp must be prolonged, and that the symptoms not infrequently developed long after the removal of the cause.

In a certain number of cases there is a history of rheumatic fever, and this, as has been pointed out, probably accounts for most of the instances in which valvular disease is met with in association with rheumatoid arthritis. In some instances it is probable that an acute attack of rheumatoid arthritis has been mistaken for true rheumatism.

In the series of cases I find mention of previous attacks of rheumatic fever in eight. In four cases morbus cordis

<sup>1</sup> 'Dublin Journal of Med. Sci.,' 1885, i, 490.

<sup>2</sup> 'British Medical Journal,' Aug. 2nd, 1884.

is noted, and of these patients two had had rheumatic fever, and two, aged sixty-three and forty-six respectively, had aortic murmurs only.

Lastly, any depressant cause, such as a period of ill-health, overwork, or loss of blood, appears to be able to bring on the disease.

In his work on 'Diseases of the Nervous System,' Dr. Gowers mentions mental distress and anxiety, severe fright, exposure to wet, cold, and accidents such as spinal concussion or injury to one limb, as causes of progressive muscular atrophy. This resemblance between the causes of progressive muscular atrophy and those of rheumatoid arthritis is most interesting, for some observers have thought that spinal arthropathies result from lesions of the anterior horns of the grey matter, and arthropathies have been observed in connection with progressive muscular atrophy.

Leaving the subject of the causation, I will now pass on to speak of the distribution of the lesions in rheumatoid arthritis.

Dr. Senator<sup>1</sup> speaks of the symmetrical onset and progress of the disease as being hardly explicable, except upon the supposition of a central cause situated in the nervous system. That symmetry is one of the most marked characteristics of rheumatoid arthritis is a fact which is so generally recognised that it requires no proof. Sir James Paget and Dr. William Budd, in their papers on 'Symmetry in Disease' read before this Society many years ago, drew largely upon rheumatoid arthritis for their examples, and in some individual instances this peculiarity is extraordinarily well marked, as is shown by the following cases.

A man, æt. 32, had the middle joints of both index, ring, and middle fingers affected.

In a woman, æt. 64, the disease began in the middle joints of both middle fingers.

<sup>1</sup> Ziemssen's 'Handbuch der Speciellen Pathologie und Therapie,' Bd. xiii, S. 143.

A woman, æt. 49, had the end joints of the index, middle, and little fingers affected.

In a woman, æt. 36, the middle joint of the right middle finger was first attacked, and then the corresponding joint of the left hand.

A woman, æt. 52, had the knuckles of both index and middle fingers affected, as well as the metacarpo-phalangeal and interphalangeal joints of both thumbs.

A man, æt. 49, had the end joints of the index, middle, and little fingers attacked.

It is a curious fact that the joints of the ring finger appear to enjoy greater immunity than those of the other fingers. My father tells me that he has also noticed this.

Frequently the corresponding joints are not simultaneously attacked, but it is the exception to find a joint remaining long unaffected when the corresponding joint has become diseased, unless the case be a monarticular one.

In a considerable number of cases all the joints of one side of the body are worse than those on the other, and in rare instances, as the following, the disease is entirely unilateral. A woman, æt. 42, had the right elbow, right knee, right side of the neck, and right temporo-maxillary joint affected.

In some such cases Dr. Ord has found ovarian tenderness on the corresponding side.

Dr. Charcot has stated that the disease usually tends to spread from the periphery towards the trunk. That there is such a tendency I am convinced, but this point is much more difficult to demonstrate than the preceding, and in many instances there is certainly no such definite order of progression.

In the case of the lady mentioned at the beginning of this paper the order of the invasion of the joints accorded entirely with this view, the feet, knees, hands, elbows, shoulders, and hips being attacked in succession.

In order to ascertain the usual mode of progression of the disease, I have constructed a table showing the

NOTES

number of times that each joint was attacked in the series of 500 cases. As the series contains cases in all stages of the disease, the order of frequency should correspond to the order of invasion; since the joints which are usually attacked late will, as a rule, be affected in advanced cases only, whereas those which are attacked early will be more frequently affected.

Joints affected.	Total.	Percentage.
Hands . . . .	430	86
Knees . . . .	303	60·6
Feet . . . .	172	34·4
Ankles . . . .	137	27·4
Wrists . . . .	133	26·6
Shoulders . . . .	125	25
Elbows . . . .	125	25
Hips . . . .	73	14·6
Temporo-maxillary . . . .	125	25
Neck . . . .	178	35·6
Back . . . .	15	3·0
Sterno-clavicular . . . .	2	0·4

The involvement of shoulders and elbows is equal, so that with the notable exception of the knees, this table shows a tendency of the disease to advance up the limbs from the periphery.

That there is no element of chance in the position of knee implication in the table is shown by the fact that it occupies the same position in each hundred cases considered separately.

	I.	II.	III.	IV.	V.
Hands	86	85	85	86	88
Knees	68	65	59	55	68

In 252, or rather more than half the cases, the disease began in the hands, in 64 in the knees, in 28 in the feet, so the knees must be regarded as especially subject to rheumatoid arthritis.

In the case of the neck and jaw the affection usually

amounted to stiffness only, but although the liability to jaw affection and spondylitis has been long recognised, and has been especially insisted upon by Sir Alfred Garrod, I think that the above statistics throw a new light upon the frequency of their occurrence.

When the male and female cases are separated the order of frequency is somewhat modified, owing to the greater frequency of monarticular cases among men.

With a view to the comparison of the distribution of the joint lesions with that of nervous arthropathies, I have collected some cases of the latter. Such cases are, however, with the exception of the arthropathies of tabes dorsalis, few and scattered.

It is well known that in tabes the joints attacked are usually one or two of the larger ones, and this agrees on the whole with what is found in the case of other more or less local spinal lesions. On the other hand, in cases diagnosed as concussion of the spine, the arthropathies, when they occur, tend to be distributed very much in the same way as the lesions of rheumatoid arthritis. This is what we should expect, seeing that in concussion of the spine we have a cause more nearly approaching those which tend to produce rheumatoid arthritis than any other in connection with which arthropathies have been observed.

I have found four recorded cases of concussion of the spine followed by joint troubles.

The first of these was reported by Dr. J. K. Mitchell<sup>1</sup> in 1833. The patient was a medical man who was thrown from his carriage, alighting upon the back of his neck and shoulder; he was instantly paralysed, partly in the arms, wholly in the legs. On the following morning there was swelling, heat, and redness of the hands and wrists with much pain. The pain was always abated by remedies applied to the affected part of the spine, and aggravated by pressure or friction there. The patient had previously had rheumatism. He recovered.

<sup>1</sup> 'American Journal of Medical Science,' 1833, vol. xii, p. 360.

The second case, which was reported by Sir William Gull,<sup>1</sup> is that of a man, æt. 38, whose hands, feet, wrists, and ankles were affected. He had partial paraplegia and anæsthesia of the lower limbs, and the sphincters were weak. The joints were reddened as in gout. The joint affection lasted for months. Here, also, the diagnosis was concussion of the spine.

In the third case, which is reported by Mr. McArdle,<sup>2</sup> of Dublin, the patient, a man, æt. 60, who had no hereditary tendency to joint trouble, fell backwards on a flagged floor. Here the knees, hips, and ankles were affected. There was paraplegia affecting the lower extremities. The patient made a gradual but complete recovery.

The last case was recently reported by Mr. Wherry,<sup>3</sup> of Cambridge. The patient was a man, æt. 34, who fell from a haystack and struck his left side. The diagnosis was "concussion of the spine?" When brought to the hospital he was in a semi-conscious condition but answered questions when roused, and it was some time before his mental powers were fully restored. In addition to the joint troubles there was sweating of the left side of the forehead and herpes over the left supraorbital nerve. The joint troubles began about a week after the accident, and the joints affected were the wrist, those of the first and second fingers, the ankles, and probably the hips and knees slightly.

Mr. Wherry states, "There was not any difference between the appearance of the joints and that which manifests itself in rheumatism or rheumatic gout." And, again, "The small joints, wrists and ankles, were most affected, the left wrist acutely." As in the three other cases the joints soon recovered completely under treatment directed to the spinal trouble.

I do not mean to imply that these cases are of the nature of acute rheumatoid arthritis, but merely to point out that

<sup>1</sup> Guy's Hospital Reports, 1858.

<sup>2</sup> 'Dublin Journal of Medical Science,' 1885, i, p. 490.

<sup>3</sup> 'Lancet,' 1886, i, p. 643.



in such spinal lesions the distribution of the joint troubles resembles that met with in that disease, there being the same tendency to symmetry, and to the affection of the small peripheral joints.

Although in this paper I have covered but a small portion of the subject, I think that the statistics which have been brought forward tend, on the whole, to support some such view of the etiology of rheumatoid arthritis as has been suggested by Dr. Ord.

We may adopt, at least as a working hypothesis, the view that the cases which we include under that name are cases of trophic joint changes due to some disturbance of the central nervous system. That such disturbances may be produced by a variety of causes, directly by mental anxiety or shock, or by the action of damp, cold, or indirectly by uterine disorders or local injuries. Lastly, we may suppose that in those who inherit the arthritic diathesis such influences would more readily lead to the production of joint lesions than in those who have no such hereditary tendency.

We must of course look to morbid anatomy to play the chief part in the further elucidation of the problem, but the study of the clinical aspects of the disease is also of great importance.

I, at least, believe that the lines of argument above discussed are those which must be followed up if the nervous theory of rheumatoid arthritis is ever to be established upon a firm basis.

To the associated muscular and cutaneous dystrophies I attach but slight importance in this connection, believing them to be only secondary to the joint lesions.

Dr. Charcot has described cases in which trifling injuries to joints, followed by slight arthritis, have led, by reflex irritation as he believes, to wasting of the muscles of the limb, and increase of the tendon reflexes, and it has been my good fortune to see two such cases under his care at the Salpêtrière.

That muscular wasting may result from any form of

joint lesion has long been known, and in rheumatoid arthritis, in which disease we have the most chronic and persistent joint lesions of all, we should naturally expect to meet with secondary trophic changes in their most pronounced form.

The recent observations of MM. Pitres and Vaillard,<sup>1</sup> who have met with changes in the peripheral nerves in this disease, suggest that such changes may perhaps play an important part in the production of secondary nervous phenomena.

<sup>1</sup> 'Bull. de la Soc. Biolog.,' July, 1886.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 311.)



A C A S E  
OF  
NEUROTOMY OF THE THIRD DIVISION  
OF THE FIFTH NERVE  
WHERE IT ISSUES FROM THE FORAMEN OVALE.

BY  
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Received April 19th—Read December 13th, 1887.

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THE fifth cranial nerve is notorious for the frequency with which its various branches are affected with neuralgia, and there can be little doubt, as Hyrtl points out, that the narrow bony passages through which the various branches of this nerve pass, explain this predisposition. To this we must add the exposed situation of its terminal branches and the intimate relation of some portions of its ramifications to the teeth and mouth.

Most of the branches belonging to the fifth nerve have been divided designedly by surgeons, either in superficial situations as at the supra- and infra-orbital foramina, or in such recesses as the speno-maxillary fossa and descending palatine canal. The speno-palatine ganglion has been attacked from its orbital aspect, as well as by hot wires

passed up the posterior palatine foramen. The lingual and inferior dental nerves have been divided from within the mouth, whilst the inferior dental nerve has been divided through a trephine hole in the inferior maxilla.

The present paper contains the details of a case in which I divided the third division of the fifth nerve at the spot where it issues from the foramen ovale.

George R—, æt. 64, came under my care in the Middlesex Hospital in August, 1886. He was suffering from a mass of epithelial cancer implicating the right side of his mouth, far too extensive to admit of removal. In addition to the suffering usually caused by this disease, his life was rendered more intolerable by sudden spasmodic seizures of pain, occurring sometimes as frequently as three times in five minutes. Indeed, the agony of this man was very distressing. Local applications of belladonna, aconitia, and subcutaneous injections of morphia were freely used. Although these anodynes removed the local discomfort, they were perfectly useless in subduing the spasmodic seizures.

An examination of the growth and the distribution of the pain led me to believe that, in all probability, the third division of the fifth nerve was implicated in the cancerous mass, and the only method of affording the desired relief would be to cut down upon the nerve as it issued from the cranium. The risks and difficulties of the proceeding were carefully considered and explained to the patient, but his only answer was, "Do anything to ease my pain."

On September 11th I raised up a flap from the right cheek immediately below the zygoma, and exposed the condyle and coronoid process of the lower jaw. With a trephine a circular portion of the ramus of the jaw was removed, involving one half of the neck of the condyle, and limited above by the sigmoid notch. This enabled me to pass my finger above the external pterygoid muscle, and, aided with a pair of forceps, the spheno-maxillary fissure was reached and the pulsations of the internal

maxillary artery distinctly felt. The cavity was now so deep that it accommodated the whole length of my index finger, and so dark that even by the aid of a benzoline reflecting lamp the nerve could not be recognised. With the finger the foramen ovale could easily be distinguished, and likewise the spine of the sphenoid bone. With a blunt-pointed bistoury the structures emerging from the foramen ovale were freely divided. Up to this point the hæmorrhage had been insignificant. On re-introducing the finger a tense band could be felt, and fearing it might be a branch of the nerve still uncut, it was divided. The result was a free and sudden gush of bright arterial blood. The cavity was too deep to admit of the hæmorrhage being controlled in the usual manner, so the forceps were abandoned and the wound plugged with a sponge, and the patient sent into the ward.

In the morning oozing still continued, and it was then deemed advisable to again attempt to find the artery. Chloroform was given, and on removing the sponges the blood poured out so freely that everyone thought the carotid was injured. The cavity was quickly plugged with a sponge, and I drew the man down in the bed, and there and then, tied the common carotid. This checked the bleeding, but still there was enough to make me feel uncomfortable. The wound in the cheek was therefore plugged with pieces of sponge sprinkled freely with tannic acid.

Next morning the patient was very comfortable, and stated that he was so free from pain, and to use his own expression, he "felt in heaven" compared to his previous condition.

On testing the sensibility of the affected side of the face I had the satisfaction of finding that the parts corresponding to the distribution of the lingual, buccal, and inferior dental nerves were anæsthetic. The region corresponding to the supply of the auriculo-temporal was not thoroughly insensible, but only benumbed. This led me to believe that the roots of the nerve had not been com-

pletely divided, and from an anatomical standpoint it can be readily understood that their escape is explained by the fact that they lie very close to the skull, and embrace the middle meningeal artery. The skin corresponding to the lower border of the inferior maxilla was only benumbed, and this is readily explained by the circumstance that, the transverse cervical nerve, from the superficial cervical plexus, frequently supplies the skin in this situation.

From the time of the operation to the day of his death my patient's condition was one of comparative comfort, and he never had another spasmodic seizure. The wound in the cheek slowly granulated and healed. The wound in the neck healed by first intention, but fourteen days later a small abscess formed over the ligature, which was of chromicized gut. This left for one month a sinus, but eventually it cicatrized soundly.

After the operation, the cancer in the cheek made very slow progress, and instead of frequent losses of blood from the mouth, the patient only saw streaks of blood at rare intervals.

Four months after the operation slight pain returned, and this I attributed to further extension of the growth; but on re-testing the tactile sensibility I suspected that the nerve ends were uniting. About a week later this suspicion was confirmed, for dull sensibility existed over the temples and cheek. This gradually increased, and at last appeared in the tip of the tongue, but never became very marked. With increased sensibility came also renewed pain, but no spasmodic seizures, and the aching pain was always easily controlled by belladonna applied externally, and at night by a morphia injection given to procure sleep.

The man lived on in comparative comfort until April 5th, 1887, eight months after the operation. During the last six weeks of his illness the cancer invaded the neck, and pressing on the cervical nerves rendered the more frequent use of morphia injections necessary. It also projected into the pharynx, and rendered deglutition

difficult, but the application of a 4 per cent. solution of cocaine overcame this trouble.

Notwithstanding all that had been done for this man, his friends would not allow me the parts I wanted, and it was with extreme difficulty that consent to examine the head could be obtained.

The Gasserian ganglion and the parts in the immediate neighbourhood were removed, and the right common carotid artery.

An examination of the parts at the foramen ovale was interesting. The nerve on quitting the skull immediately entered a mass of connective tissue in which was embedded the internal maxillary artery. On tracing this vessel it suddenly terminated in a *cul-de-sac*, its obliterated end being continuous with the connective tissue surrounding the nerve. The internal carotid artery was intact. It was impossible to distinguish the various fasciculi and trunks of the nerves with the naked eye. By cutting sections of the nerve and examining them microscopically, many cut ends of nerves were visible, most of which had undergone degenerative changes. Other nerves, to all appearances healthy, were easily made out, but the nerve-trunks, instead of being regularly arranged, were mixed in confusion.

With regard to the ligatured artery. The common carotid had been ligatured about an inch from the innominate. Above this point it was represented as a fibrous cord for half an inch, to which the vagus was firmly united. The artery above this spot was filled with firm clot, which extended two inches into the external and half an inch into the internal carotid trunk.

The above case seems worthy of record, partly on account of its novelty and partly on account of the difficulty encountered in the operation, but chiefly for the immense relief it afforded the patient.

Should I again attempt the operation I would open widely the mouth so as to get more room under the zygoma (this was impossible in the present case on account of the



tumour). Further, I should take the precaution to secure the internal maxillary artery as a preliminary, and aided by an electric lamp endeavour to cut a piece from the nerve instead of merely dividing it.

Possibly, however, many will regard the procedure as one scarcely within the sphere of legitimate surgical enterprise, but on the present occasion it was undertaken with the most earnest desire to relieve the patient's intolerable agony.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 316.)

A CASE  
OF  
OCCLUSION OF THE LEFT BRONCHUS BY  
A METAL PENCIL CAP,  
AND ITS REMOVAL BY TRACHEOTOMY.

BY  
W. B. CHEADLE, M.D., F.R.C.P.,  
AND  
THOMAS SMITH, F.R.C.S.

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Received September 8th, 1887—Read January 10th, 1888.

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*History of the case previous to the operation, by*  
DR. CHEADLE.

THE patient was a little girl nine years old, the daughter of Mr. F. Mivart, and at school at Blackheath. On January 30th, 1887, she was sucking the end of her pencil while playing over her writing, when the loose cap used for covering and protecting the point became detached, and, by a sudden inspiration, was carried into the throat. Urgent choking and dyspnoea followed, the child's face grew livid, and she was on the point of suffocation. Dr. Creed, of Greenwich (to whom and to Mr. Mivart and Mr. Tegart, I am indebted for notes of the first phase of the case), was immediately sent for, but by the time he arrived the dyspnoea had considerably lessened; nothing could be seen in the throat, or felt there on exploration with the finger. The child still

complained of something sticking in her throat, and pointed to the middle of her neck as the place where it lodged.

A probang was passed down the œsophagus, and this appeared to give relief; but there was still great pain in the chest, and frequent cough of some violence. On physical examination, however, no change of importance was discovered. The pencil cap was believed to have entered the stomach, and its appearance looked for in the fæces. The pain in the chest gradually subsided, but the cough remained, and was observed to be always aggravated by any movement.

On February 3rd, four days after the accident, Mr. Mivart examined the child with Dr. Creed and Mr. Tegart. Although she seemed fairly well, the continuance of cough, although this was not severe, excited some suspicion and the chest was again examined. Partial dulness was now found over the left side behind, and imperfect entry of air there, especially over the middle portion. There was dulness at the left front also, although this was less marked than at the back, and the entry of air into the apex of the lung was noted as deficient. When seen again four days later still the general condition was much the same, but the dulness and want of respiratory sound on the left side had increased. Mr. Mivart also observed that the tympanitic percussion-note of stomach resonance extended upwards nearly to the level of the left nipple.

At this juncture the child was brought to London, and I was asked by Mr. Mivart to see her in consultation with Mr. Tegart and himself.

On stripping the chest two things were at once obvious viz. first, that the left side was almost motionless in respiration; and secondly, that it was smaller than the right. The heart's apex was under the fifth rib at the lower edge of the nipple, in a line with it, *i. e.* a little above and outside the normal position.

On percussing the chest the *left* side was found almost uniformly dull, and vocal vibration was impaired as com-

pared with the right side. The want of resonance was very marked but not absolute. Exceptions to this general impairment of resonance were a small area about the size of a crown piece over the second left space in front, which gave almost normal lung resonance, and the lower portion in front, below the nipple line. In the latter region this was due to the tympanitic note of the stomach, which extended upwards to the level of the nipple. On listening with the stethoscope, hardly any respiratory sound could be detected on the left side, either in front or behind. The silence was almost complete even on forced inspiration. Distant breathing only could be heard, faint and bronchial. The bronchial character of the respiration was most notable over the spine of the left scapula. No wheezing or whistling sound was audible, indicating partial passage of air, and there were no râles. Over the small, more resonant area at the second left space, some natural respiratory murmur could be heard. On the *right* side the percussion-note was fully resonant, the breathing somewhat loud and exaggerated in volume, in remarkable contrast to the feeble, barely audible, bronchial respiration on the left.

The *respirations* numbered 30 in the minute; they were quiet, easy, unforced.

The *pulse* was 92, of natural beat and character. The *temperature* was  $97^{\circ}$  in the axilla, in the mouth  $97.8^{\circ}$ . This had been taken regularly, and had never at any time been found as high as  $98^{\circ}$  in the mouth.

Now and again the child was harassed by a short hacking cough, and this was observed to be always brought on by exertion, and especially by going upstairs. When she was quietly at rest the cough subsided. There was no dyspnoea or distress of any kind connected with breathing, except the cough.

It was quite clear from the physical signs that the left lung was almost completely collapsed. The retraction of the side, the absence of movement, the rising of the stomach to the level of the nipple, the displacement of

the heart's apex upwards and to the left, showing extreme contraction of the lung, together with the impaired resonance and vibration, and almost entire absence of respiratory sound, were conclusive on this point. The history of the case left little question that this general collapse was due to the occlusion of the bronchus by the foreign body. The pencil cap, of which we obtained an exact counterpart, was a tube nearly an inch long, closed



FIG. 1.—Sketch of the pencil-cap impacted (actual size).

at one end, open at another, and appeared to be of just sufficient calibre to enter into the bronchus easily, and yet fit it pretty closely; while in length it would correspond very nearly with the lower portion of the bronchus, where the main branches are given off.

The physical signs thus threw light upon the exact position of this foreign body. The fact that a certain amount of air passed in and out of a portion of the upper lobe, evidenced by respiratory sound and resonance in a limited area of the apex in front, seemed to prove that the cap had passed on to the extreme end of the left bronchus and had thus left the first branch, viz. that which leads to the upper lobe, partially patent, allowing the air to enter this position of the lung to a limited extent, the passage to the rest being absolutely occluded. The position of the foreign body is shown in the diagram, adapted from one kindly furnished me by Mr. Rickman Godlee, and also in the specimen of lung and bronchi with the foreign body *in situ*, kindly prepared by Dr. Penrose, which serves admirably to illustrate the case. I had no doubt as to the impaction of the foreign body in the left bronchus therefore, and that its removal by tracheotomy should be attempted as soon as possible, before serious inflammatory changes were set up in the surrounding tissues.

It was agreed that Mr. Thomas Smith and Dr. Broadbent should first be asked to see the case independently, with a view to the verification of the exact diagnosis, and that we should then hold a consultation to determine finally the question of tracheotomy.

This was done with the result of confirming the diagnosis as to the occlusion of the bronchus by the pencil cap, and its position at the extremity of the main tube. Dr. Broadbent pointed out the probability that the rounded closed end of the cap would pass first into the bronchus. It was obvious that this end would lie towards the throat

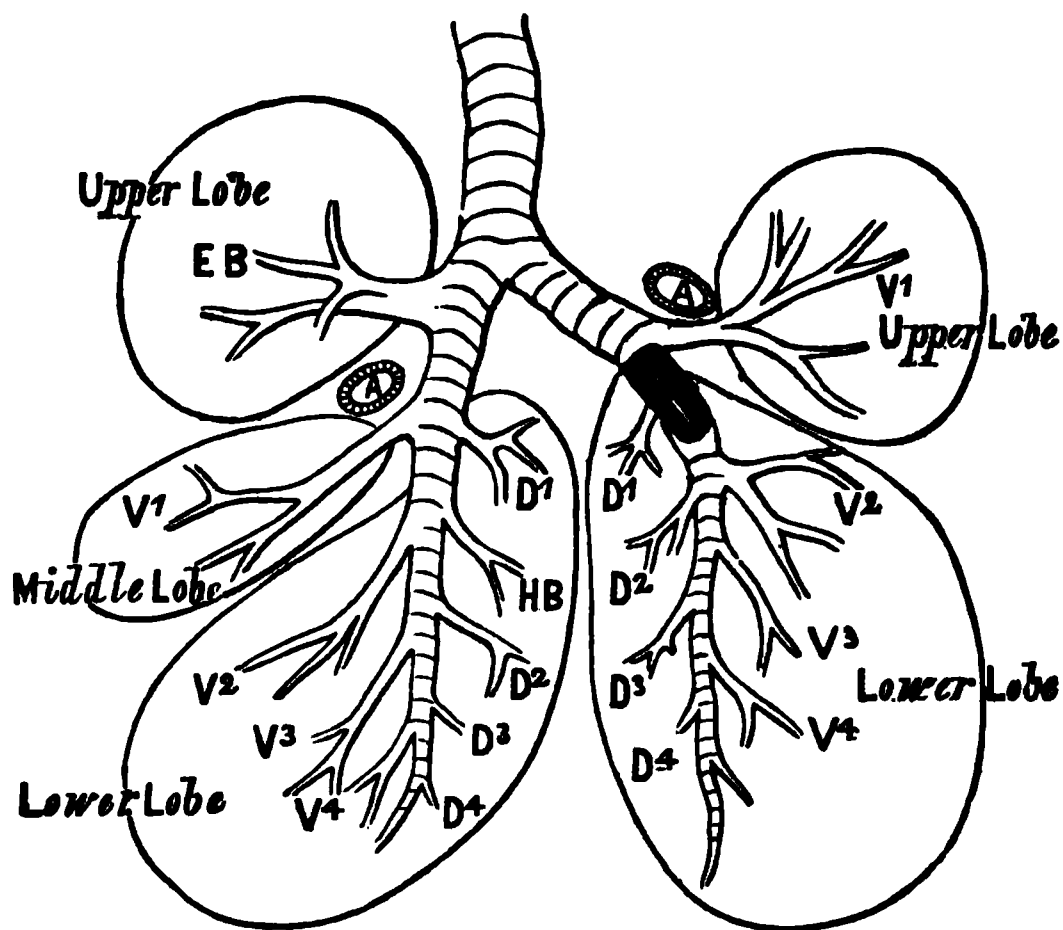


FIG. 2.—Diagram of the bronchi, showing the position of the foreign body at the extremity of the left bronchus, beyond the origin of the branch to the upper lobe, which was thus left free. [Adapted from a diagram modified by Mr. Rickman Godlee from one by Dr. Aeby, of Berne.]

as the pencil was being sucked, and it would be less liable to trip or catch in transit. There was therefore a reasonable expectation that the open end would be found uppermost, an important consideration in regard to the prospect of the success of the operation. For the open extremity of the tube would be much more easily laid hold of

than the closed rounded end, which would clearly present great difficulties in the way of firm grip by the forceps.

The operation was successfully performed by Mr. T. Smith, assisted by Mr. Howard Marsh and Mr. Holmes, on the 17th of February.

*Account of the operation by Mr. T. SMITH, with remarks.*

The specimen furnished us of the foreign body was cylindrical, closed, and rounded at one end, open at the other, with a ring-like depression about the middle of the tube inside. It was  $\frac{15}{16}$ ths of an inch long and  $\frac{5}{16}$ ths in diameter. As there was no certainty that the open end lay uppermost in the bronchus, provision was made for seizing whichever end might present.

In case the open end lay uppermost Mr. Mayer provided me with forceps having an external grip, such as are used for intubation of the larynx ; these placed within the pencil cap held it firmly when their blades were expanded, and they were suitably curved for reaching the bronchus. The closed rounded end of the foreign body could not have been grasped by any forceps from above. I therefore provided myself with means for meeting this difficulty should it occur.

In the operation, after stopping all bleeding and dividing the isthmus of the thyroid body between two ligatures, the trachea was freely opened and the edges of the tracheal wound were temporarily attached to the margins of the skin-wound by one or two silk sutures. A long curved slender probe was passed into the bronchus, and at once detected the foreign body in the position assigned to it by physical examination, and from the sensation communicated to the probe it was judged that the open end of the pencil cap lay uppermost.

The intubation forceps were introduced and pressed firmly against the foreign body, so as to get a good hold of its interior, and the blades being expanded it was at once extracted. The tracheal sutures being removed the

wound was dressed with carbolic oiled lint and left to granulate.

I would venture to recommend the plan of attaching the edges of the tracheal wound to the skin while searching for foreign bodies in the air-passages below; it keeps the trachea widely open throughout the operation, which is a great help to the surgeon and a source of safety to the patient.

*Subsequent history of the case, with observations by*  
DR. CHEADLE.

When the child was examined the same evening, four hours after the operation, air was found to be entering the left lung very fully, with harsh expiratory sound, except the upper part behind; here there was still comparative silence. There were no râles audible anywhere. Resonance had improved in marked degree. The temperature was still only  $98^{\circ}$  and the respirations 30, but the pulse had risen to 120. The child was quiet and seemed comfortable. On the following morning, however, February 18th, there was a sudden access of fever. The temperature had risen from its previous subnormal level to  $104^{\circ}$ , the pulse to 156, the respirations to 44, and the child had vomited twice. Minute examination of the chest disclosed no cause for the febrile disturbance. Air was found to be entering freely except at the upper part of the left lung behind. Respiratory sound was harsh, but there was no approach to bronchial breathing, and there were no râles or added sounds anywhere. Resonance was still deficient. By 5 p.m. the same day these ominous symptoms began to abate, the pulse had fallen to 130, the temperature to  $101^{\circ}$ , the respirations to 40.

Next morning, February 19th, the temperature had fallen still further, viz. to  $100^{\circ}$ , the pulse 116, the respirations 30. There had been no more sickness. Auscultation showed good respiratory murmur all over the left



lung, the upper part now filling with air like the rest ; in fulness of breath-sound and expansion this side was still deficient as compared with the right. The only abnormal sounds were some harshness of respiratory murmur and a few medium-sized moist râles at the second and third left cartilages, *i. e.* about the position of the termination of the left bronchus and the commencement of its first branches. The further expansion of the lower lobe proceeded slowly now ; the respiratory sound was less full there, and the stomach-note still reached to the sixth rib. The pulse, respirations, and temperature continued to fall, so that by February 24th, *i. e.* seven days after the operation, these were 92, 24, and  $98.6^{\circ}$  respectively. Some bronchial respiration was now for the first time made out at the posterior axillary line, at the level of the angle of the scapula on the left side, with fine moist crepitations as of freshly expanding lung. The resonance over this point was still slightly deficient.

During the following week these abnormal signs gradually declined and disappeared by March 1st. The temperature again fell to subnormal, and remained between  $97^{\circ}$  and  $98^{\circ}$ , but never reaching  $98^{\circ}$  up to the time of the latest observation on March 8th, nineteen days after the operation, thirty-six after the accident.

A final examination of the chest at this date showed normal respiration everywhere restored on the left side, except that it was slightly rough and generally rather deficient as compared with the right. Resonance and expansion were also below the standard of the right side, but the stomach-note of resonance had dropped to the seventh space, and the apex-beat of the heart to the normal position in the fifth. Measurement of the two sides of the chest still gave a difference of half an inch in favour of the right, which was twelve and three quarters inches as compared with twelve and a half on the left.

There seems to be a general acceptance of the statement that foreign bodies entering the trachea almost invariably pass into the right bronchus, and an anatomical

reason has been found for this in the larger size of the right tube, and the fact, first pointed out by Dr. Goodall, of Dublin, that the septum between the two bronchi is placed to the *left* of the middle line, thus making the lumen of the *right* more in the direct line of the axis of the trachea than the left.

In 1855 Dr. Hughes published in the 'Dublin Quarterly Journal' a case of impaction of fish-bone, which he claimed to be singular, as the first recorded in which the foreign body had passed into the *left* bronchus.

The statistics collected by Bourdillat ('Gazette de Paris,' 1868) show twenty-six cases of impaction in the right bronchus to fifteen in the left. Professor Gross, of Philadelphia ('Practical Treatise on Foreign Bodies in the Air Passages,' 1854), out of sixty-three cases found thirteen certainly and eleven probably in the right to four certainly and four probably in the left, or twenty-four to eight. And yet out of the thirty-four cases which I find recorded in the English journals, in the thirty in which the position of the foreign body is stated, in sixteen it was in the *left* bronchus as compared with fourteen in the *right*. In three cases which came under my own observation it was in the left in every one, making in all nineteen left to fourteen right. This at least shows that, as Reigel<sup>1</sup> affirms, the entry of a foreign body into the left bronchus is by no means so infrequent as might appear from the reports of many authors.

The physical signs observed in this case do not correspond exactly with those accepted by authority as typical in such conditions. It will be remembered that the left side of the chest was noted to be markedly deficient in resonance on percussion. This is exactly what might be expected, what I have found in a similar case, and what I believe always occurs in extensive collapse of the lung. Yet I find it stated by Reigel that on the affected side there is "full and clear sound on percussion which all observers admit differs in nothing from the normal

<sup>1</sup> 'Ziemssen's Cyclopædia.'

sound.” And, again, in the recent work of the late Dr. Fagge the percussion sound is said to be normal. This is, I am sure, erroneous. In another case of the kind which came under my observation, in which a pea entered the left bronchus, causing complete obstruction, the percussion-note was distinctly dulled on the affected side. The mistake has arisen probably in this way :

1. In cases where obstruction of the main bronchus is complete, before the air is pumped out of the lung or absorbed, and collapse established, resonance is unchanged, and this is the stage which has been noted and regarded as typical. But later, as the air disappears, and the lung becomes collapsed and carnified, resonance becomes more impaired. That collapse had already commenced in the present instance in four days after the accident was evidenced by the already distinct want of resonance and breathing on the left side of the thorax, and four days later by the further increase of this and the rising of the stomach to the fifth space noted by Mr. Mivart.

2. In cases where obstruction to the entry of air *is not complete*, as when the foreign body is of a form which does not entirely close the tube to the passage of air, there may be little or no collapse ; or when the foreign body is not long enough to stop all branches when driven to the end of the bronchus, collapse may be partial and slight, and the difference of percussion-note hardly appreciable.

Again, this case is interesting since it affords further proof of the correctness of Dr. Gairdner's theory of the production of collapse of portions of lung by obstruction to the entry of air by the supplying bronchus ; and shows how complete obstruction of the main bronchus causes absolute collapse of the whole of the lung.

Another point of interest arises in connection with this question of collapse. This case proves that mere collapse is not, in itself alone, a cause of catarrhal pneumonia, or even the chief and most potent factor, as suggested by Niemeyer. The lung in this instance was very completely

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collapsed for ten or twelve days before the operation, yet no inflammatory change ensued. There was not only no rise of temperature at any time, but it was persistently subnormal. There was never any crepitation audible until after re-expansion commenced.

The condition of the temperature, of respiration, and of the circulation, deserves a moment's notice. The temperature was always subnormal, viz.  $97^{\circ}$  to  $97.6^{\circ}$ . The respirations were increased in frequency, standing at 30 with remarkable constancy, but not doubled, although one lung had to do the whole work of aeration. The pulse-rate ranged between 90 and 100, so that the number of respirations showed an increase of about one third over the normal, the pulse-rate only a quarter over the normal. Yet this was insufficient to compensate fully for the loss of oxygenating power caused by the loss of function in the closed lung, and keep the temperature up to the normal standard.

The sudden sharp burst of pyrexia after the operation is not easy of explanation. I find that a rise of  $102^{\circ}$  and  $103^{\circ}$  is recorded in two other cases on the second and third day after a similar operation for the removal of a foreign body.<sup>1</sup> In the present instance the pyrexial period was of extremely short duration, reaching its acme,  $104^{\circ}$ , in nineteen hours, subsiding in the next six hours to  $101^{\circ}$ , and it was practically at an end by the third day. Was the fever of traumatic origin? There is little to support this hypothesis. The wound did well from the first. Was it due to abortive pneumonia, of which the moist râles heard on the seventh day after the operation were the only and posthumous evidence? I think not, for such râles are constantly heard over the small bronchi when lung is re-expanding after collapse, as, for example, after tapping in pleuritic effusion, and there was never any other sign in any way suggestive of pulmonary inflammation. The pyrexia may have been due to shock and the excitement of the operation,—purely neurotic. Another possible explanation is that the fever wave was due to the dis-

<sup>1</sup> Marcus Beck, 'Lancet,' ii, 1874, p. 798. Lucas, 'Lancet,' ii, 1881, p. 874.

turbance produced by the sudden abrupt forcing into action of the function of the lung previously in abeyance. The re-entry of air would doubtless produce irritation of the bronchial mucous membrane, and might give rise to neurotic disturbance of temperature, while the oxygenation of the blood would be instantly increased, and metabolism and heat production possibly thus raised, temporarily, to excess.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 321.)

INFLUENCE  
OF  
SALICYLIC ACID AND ITS SALTS ON  
THE EXCRETION OF URIC ACID.

BY  
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IN a paper which I read before the Society in May, 1887, I drew attention to the relation of a certain form of headache to the excretion of uric acid, and showed that the headache in question coincided with a plus excretion of uric acid, and was possibly due to an excess of uric acid in the blood. And in the 'Journal of Physiology,' vol. viii, p. 211, I have narrated some experiments on the effects of acids and alkalies on the excretion of uric acid, and have mentioned that by giving acids I am now able both to cure the above form of headache and to stop the plus excretion of uric acid which accompanies it.

It may be taken then, as shown in the latter paper, that alkalies, as soda and potash, always, within certain limits, increase the excretion of uric acid, while *acids* as nitro-hydrochloric, citric, acetic, always diminish the

excretion of uric acid ; but there is one important exception to this rule in the case of salicylic acid, and as the value of this drug in rheumatism and gout is so great I propose to go at some length into its exceptional action and the results of my experiments with it as viewed in the light of my papers above referred to.

Shortly stated, the action of salicylic acid, or salicylates, is to dissociate the excretion of uric acid from its general relation to acidity, so that an increase of acidity, while there is plenty of salicylate in the blood, is not accompanied by any diminution of uric acid excretion.

The great importance of this fact may be roughly estimated by considering the *rôle* played by acids in dyspepsia, in gout, and in rheumatism ; and if, as I hope to be able to show, salicylates prevent these acids causing retention of uric acid, a large part of their action in these diseases will admit of easy explanation. Thus of gout Sir A. Garrod says ('Gout and Rheumatic Gout,' ed. iii, 245) : "When a few glasses of wine, ale, or porter quickly and invariably produce in any individual an inflammatory affection of a joint, such inflammation is of a truly gouty character." The way in which these beverages act in producing the inflammation is now, I think, sufficiently clear. I shall not go further into the ultimate pathology of rheumatism and gout but at once bring forward the results of my experiments with salicylates.

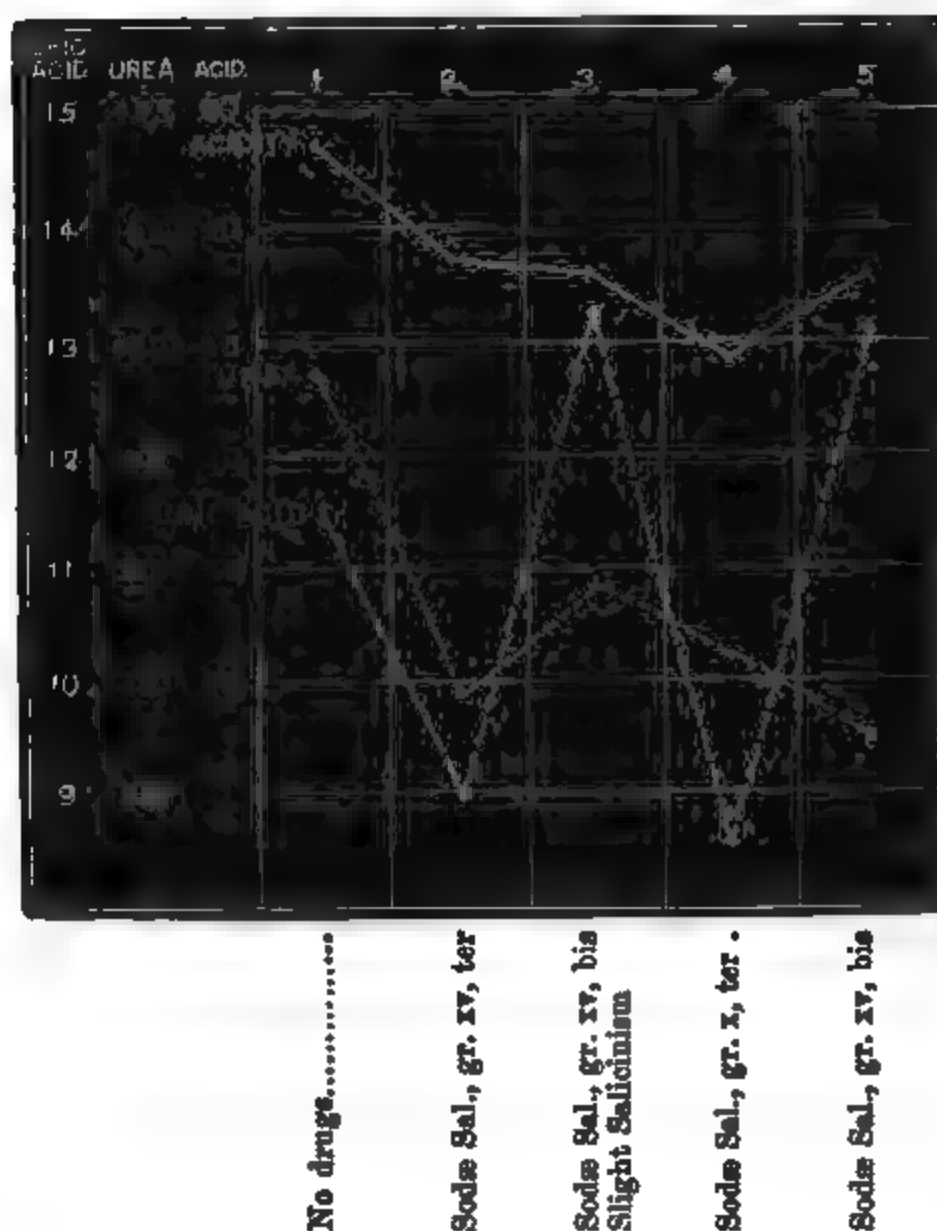
The results of one of my first experiments is given in Fig. 1.

On the first day in this figure acidity of the urine is very high (owing to drugs taken on the preceding days not shown in the figure), and uric acid is, as one would expect, below the urea, *i.e.* there is retention of uric acid due to the high acidity.

It is here assumed, for reasons given in previous papers, that the normal relation of uric acid to urea in this case is 1 to 33, and on this relative value the curves in all the figures are based.

On the second, third, fourth, and fifth days salicylate

FIG. 1.



of soda is taken in amounts varying from 30—45 gr. a day.

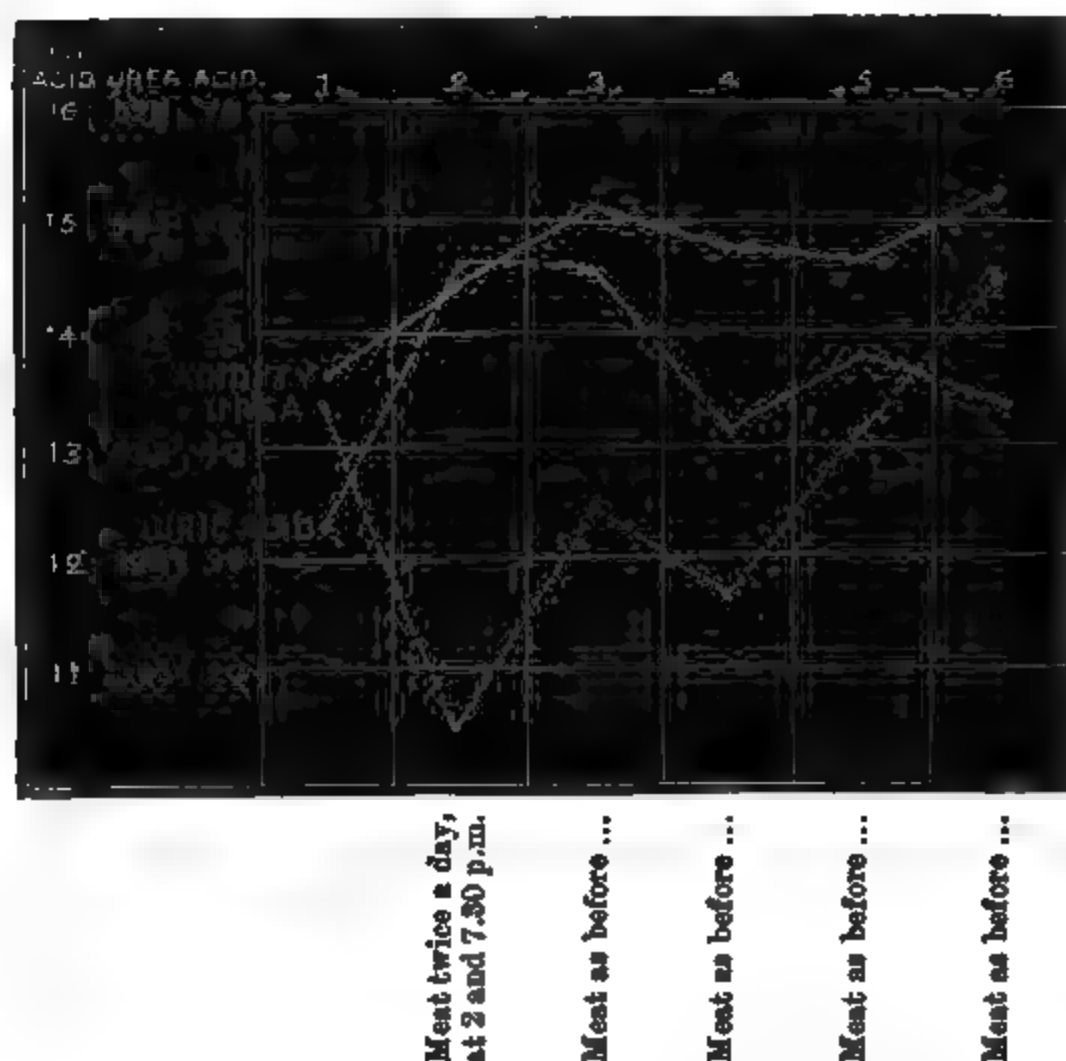
It does not appear to have any definite effect on the acidity of the urine, which rises or falls from other causes, but it is at once seen that the uric acid excretion is not influenced by the acidity in the usual way, for on the third day it rises high above the urea, while the acidity is almost the same as on the previous day, and on the fourth day uric acid actually falls below urea, though as acidity has fallen we should have expected a rise. Again, on the fifth day acidity has risen yet uric acid is not kept down by this, but actually rises also ; so that we see that



under salicylate the uric acid excretion fails to keep up its usual inverse ratio to the urinary acidity; indeed, it almost seems as if the reverse effect were produced, so that a fall of acidity corresponds with a fall of uric acid, and a rise of acidity with a rise of uric acid.

For the purpose of contrast with these salicylate of soda curves, I have given in Fig. 2 several days when no

FIG. 2.



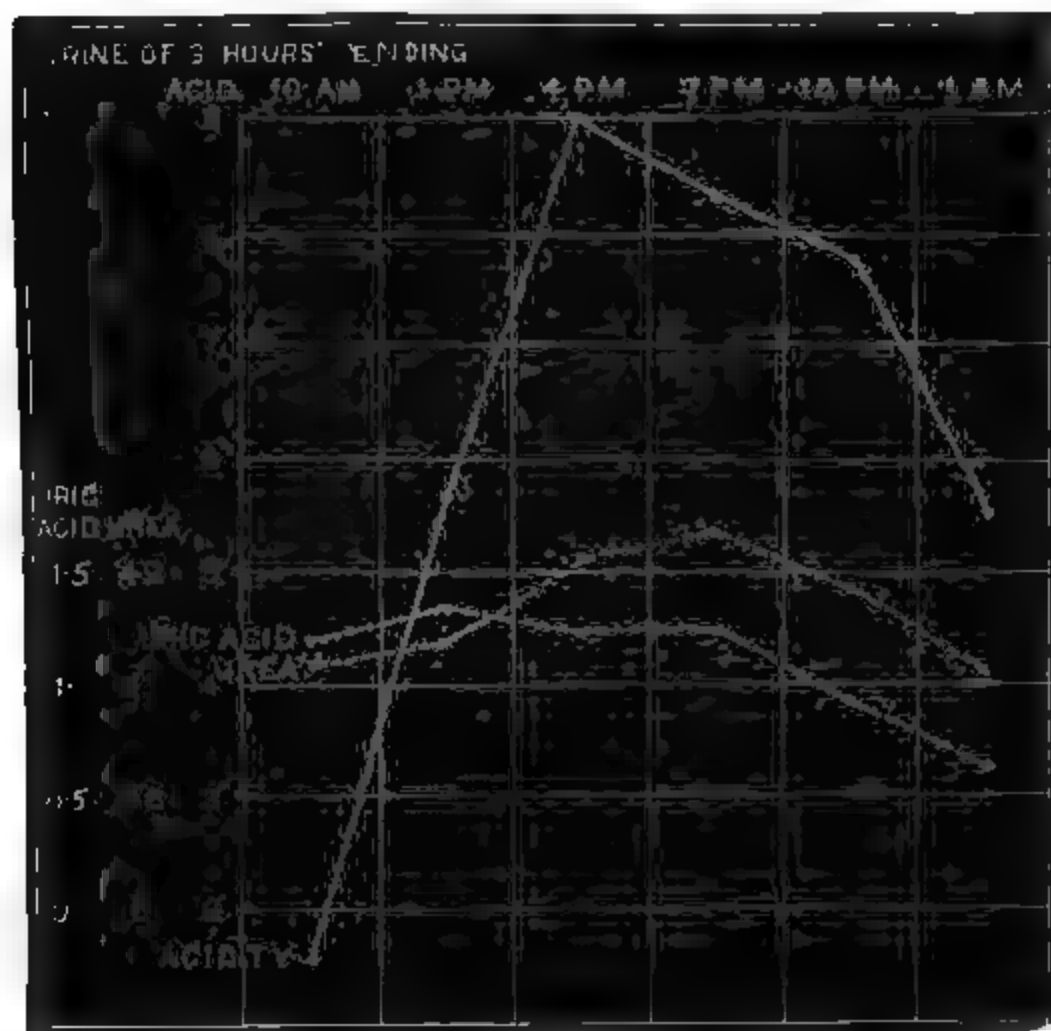
drugs were taken, so as to show the ordinary relation of uric acid to acidity. This is no picked instance but the first that comes to hand; indeed, I have very few curves of periods when no drugs were taken, and even during these curves in Fig. 2 meat was being taken from the second onwards for purposes of experiment.

Now, at first sight it might appear as if this figure and

its curves would upset all my previous results as to the intimate relation of the excretion of uric acid to acidity, for while on the second day acidity is rising, uric acid is a long way above urea, and my notes show that there was a very decided headache on that day; but on closer examination I think it will appear that this exception is more apparent than real, and that the uric acid is really influenced by the acidity in the way I have pointed out in previous papers.

On the day on which this apparent exception occurs meat was taken twice, viz. at 2.0 and 7.30 p.m., and as it is well known that meat increases acidity I take it for granted that the rise in acidity is due to the meat; but it is also well known that the great excretion of uric acid in each twenty-four hours takes place during the alkaline tide after breakfast in the morning and the few following hours. On the day in question, however, the first meal of meat was not taken till 2 p.m., and it would not have any action on acidity for one or two hours more, so that the great uric acid excretion for the day had taken place *before* the acidity rose, and so could not be affected by it. This is the explanation of several apparent exceptions to the rule, and I have often found that taking meat and wine at dinner affected acidity, but had little influence on uric acid, though their action on the uric acid excretion of next day was clear enough. In this case we see that on the third the uric acid is little more than half the distance above the urea it was on the second; on the fourth and fifth it gets still nearer to it, and on the sixth, with a further rise of acidity, it is below it. And while speaking of this matter, I will just draw attention to Fig. 3, showing the acidity, uric acid, and urea curves, as obtained from urine collected in consecutive periods of three hours from 7 a.m. one day to 1 a.m. the next. This shows clearly enough the effect of acidity on the uric acid excretion, but also shows that the greatest uric acid depression does not occur for some three hours or so after the greatest rise of acidity. So that we see that a rising

FIG. 3.



acidity takes several hours to exert an appreciable influence on uric acid excretion; and further that, if the rise in acidity is not caused till late in the day, it cannot influence the uric acid of that day's excretion to any great extent, but will act most on the alkaline tide excretion of the next day.

Now, to return to the effects of the salicylate in counteracting this usual effect of acidity on uric acid. In Fig. 4, which is consecutive to Fig. 2, one day being common to each, it will be seen that the uric acid curve, though above the urea, is clearly dependent on the acidity curve, being always further above the urea when acidity falls, and closer to it when the acidity curve rises; and this holds good up to the tenth. But on the eleventh, the first day of salicylate (taken as salicylic acid so as not in any way to diminish the acidity and so complicate

matters), there is a marked change in the relations, for acidity rises sharply, and in spite of that uric acid rose considerably. If this were due to the factors I have already spoken of as causing apparent exceptions to the rule, one would expect that the high acidity would bring down the uric acid of the next day (the twelfth); but quite the reverse is the case, for the uric acid still further rises to the large excretion of 17·4 grs., which exceeds its general relation to urea by more than 6 grs. So that I am unable to explain this extraordinary curve except as the result of the salicylate taken, and it coincides with the results obtained in previous experiments with it. It is further to be remarked that this excessive excretion of uric acid was not accompanied by any headache, which it would almost certainly have been if it had occurred apart from the action of salicylate.

On the thirteenth acidity falls and uric acid also falls, and this coincides with what we have before seen to occur under the influence of salicylate.

On the fourteenth, however, there is an exception, for acidity rises and uric acid falls and comes much nearer to the urea than it has been for some time; and on the fifteenth it is nearer still to the urea. My belief is that with the rise in acidity from the thirteenth to the fifteenth and sixteenth the uric acid would have been below the urea instead of above it, had it not been for the action of salicylate, and on the sixteenth, two days after the last dose of salicylate, it does actually fall below it.

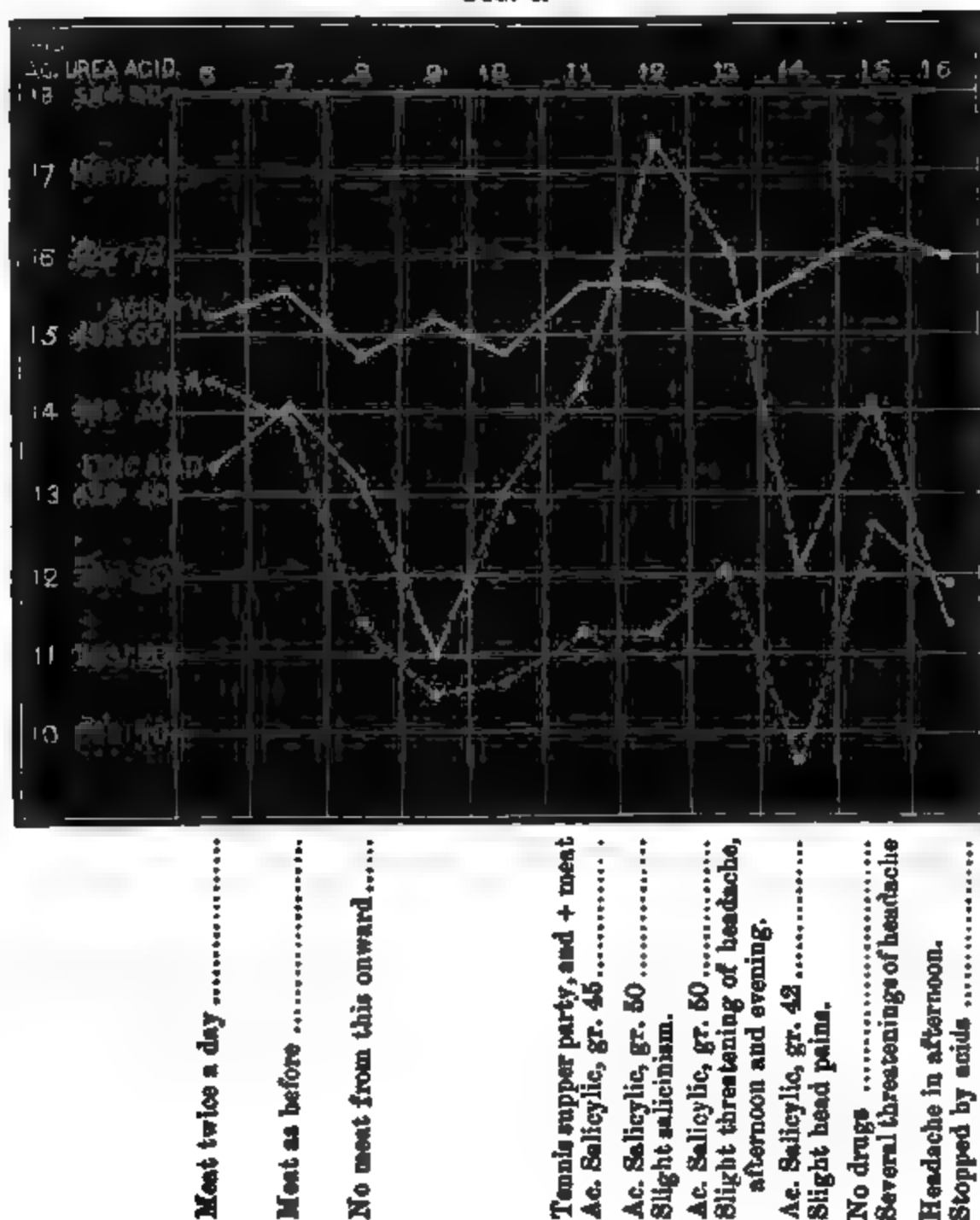
From these experiments (Figs. 1 and 4) two facts stand out clear and distinct.

(1) That under salicylate, uric acid excretion is not kept down by rise in acidity but rather the reverse; and

(2) That a large excess of uric acid excretion when it occurs under salicylate is not accompanied by headache; and there are several facts which may be placed beside this; thus, it has been shown by Dr. Brunton,<sup>1</sup> that salicylate of soda is a valuable remedy in headache, and

<sup>1</sup> 'Pharmacology, Therapeutics, and Materia Medica,' p. 559.

FIG. 4.



we know, moreover, that salicylate in one form or other relieves the pains of acute rheumatism, and prevents or relieves the pains and discomforts of many phases of gout; and I believe that this headache, for the reasons given in my previous papers on headache and uric acid excretion, is merely a symptom of so-called "quiet gout."<sup>1</sup> I consider therefore that I have good grounds for supposing

<sup>1</sup> See Mr. J. Hutchinson, 'Lancet,' Nov., 1884. p. 903.

that in these instances the salicylate prevented the headache which would otherwise have accompanied such a large excretion of uric acid.

Into the other matter, the reversal by salicylate of the usual action of acidity on the excretion of uric acid, I propose to go at some length, because though a complete explanation is not at present possible, there are several chemical facts which it is well worth while to place by the side of those I have now brought forward. Thus Professor Latham, in his 'Lectures on Rheumatism, Gout, and Diabetes'<sup>1</sup> says salicylic acid combines with glycocine and passes out in the urine as salicyluric acid, and if we may suppose that salicylic acid has the power of splitting up uric acid and combining with its glycocine so as to form salicyluric acid (just as benzoic acid does to form hippuric acid) it would not be very difficult to explain the reactions I have obtained. For what is known of the chemistry of salicyluric acid would lead us to suppose that it is more soluble than uric acid both in the tissue fluids and in slightly acid solutions, and these peculiarities would be sufficient to explain the altered relation of uric acid excretion to acidity under salicylates. I may here say that having arrived at this point with salicylate it was suggested to me by Dr. Lauder Brunton to try whether benzoates would have the same effects, and I at once performed similar experiments with these salts, and found that benzoate of ammonium, which increases acidity, diminishes uric acid excretion, while benzoate of sodium, which diminishes or does not affect acidity, allowed of an increase of uric acid excretion. The benzoates therefore appear to act merely through their effect on acidity, and not as the salicylates do; and this different effect is, I suppose, due to the fact that salicyluric acid is more soluble than hippuric.

With regard to this point, I have thought that it might be useful to give short extracts of what is said of the

<sup>1</sup> 'Brit. Med. Journ.,' 1886, p. 629, *et seq.*, and 'Lancet,' Jan., 1885, p. 1119, *et seq.*

solubilities of these acids in 'Watts's Dictionary of Chemistry.'

Thus "Uric Acid," vol. v, p. 955: "It is nearly insoluble in water, one part of it requiring 15,000 parts of cold and 1800 parts of boiling water to dissolve it; in alcohol and ether it is quite insoluble."

"Hippuric Acid," vol. iii, p. 157: "It is sparingly soluble in cold water, one part of the crystals requiring 600 parts of water at 0°. Boiling water and alcohol dissolve it readily, ether scarcely at all. Hippuric acid likewise dissolves, but very sparingly, in water containing hydrochloric acid. Hippuric acid dissolves in strong hydrochloric acid at the boiling heat, and on continuing the ebullition it is resolved into benzoic acid and glycocine."

"Salicyluric Acid," vol. v, p. 172: "It dissolves easily in boiling water and in alcohol, less easily in cold water and in ether. Heated for a short time with hydrochloric acid it dissolves and crystallizes out again unaltered, but if the boiling be continued for two to three hours the acid is resolved into salicylic acid and glycocine."

I am sorry that the statements about salicyluric acid are not more definite, but I think we may infer that both hippuric and salicyluric acids are far more soluble in water than uric acid, and are also more soluble in water containing hydrochloric acid. This latter point is not definitely stated with regard to salicyluric acid, but I think as it is more soluble in other ways than hippuric acid, and, like it, is soluble in strong hydrochloric acid, that it is probably at least as soluble as hippuric acid in weak hydrochloric acid, and that salicyluric acid is more soluble both in water and in ether, and probably in weak acids, than is hippuric acid.

If we may suppose that on giving salicylate the uric acid in the blood and tissue fluids is converted into salicyluric acid, we should have to deal with a substance which as compared with uric acid is far more soluble in those fluids, and also more soluble in slightly acid fluids;

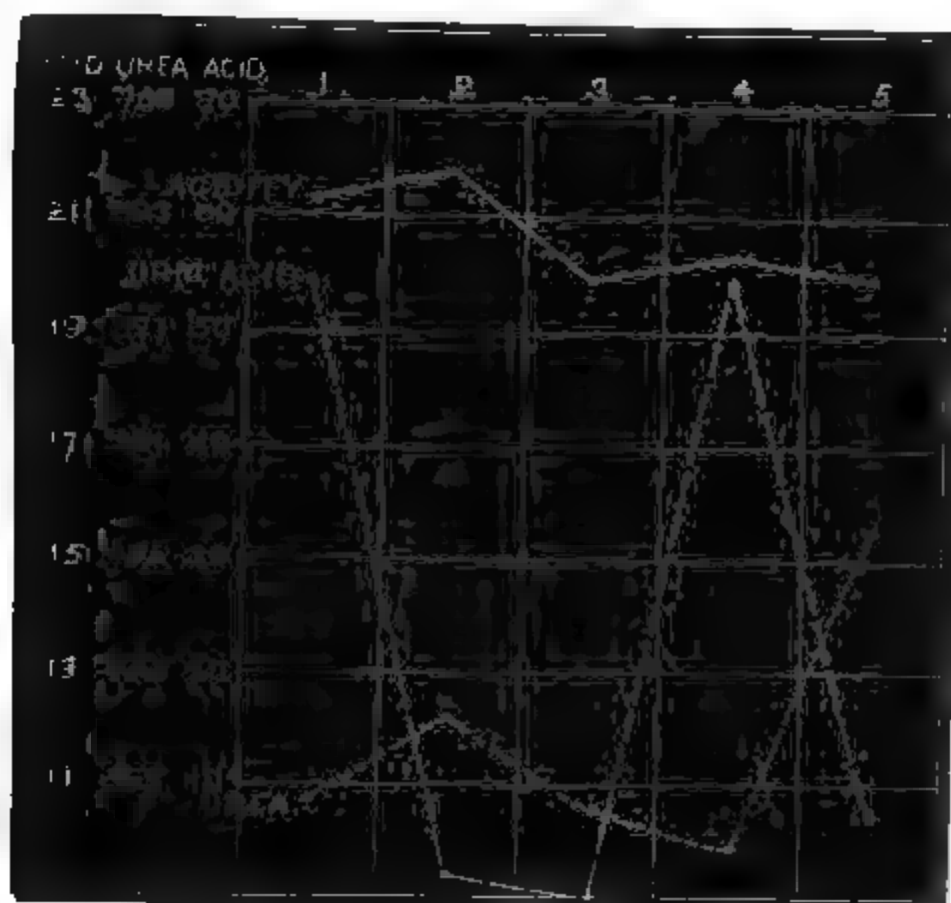
and if alteration of solubility is the explanation of the action which I have shown that acids and alkalies have on the excretion of uric acid, we have here also a simple explanation of the result I have obtained with salicylates, viz. that a rise in acidity no longer entails a fall in uric acid excretion.

And I believe that here also, just as in the case of alkalies, the salicylic acid acts on the uric acid which is supposed to be stored in the liver and spleen, and, increasing its solubility, rushes it at once into the blood and out by the kidneys. But the rush into the blood under salicylate is not accompanied by any headache, and it differs in this respect from the rush under alkalies, so that we have some grounds for the supposition that the uric acid exists in the blood in a different chemical combination (salicyluric acid) when it is washed out by salicylic acid, to what it does when washed out by alkalies. Fig. 5 will serve to illustrate several of these points. In Day I, acidity is high (probably higher than the previous day, though that was not estimated), but uric acid is far above urea because salicylic acid has been taken; on Days II, and III, uric acid is below urea, meaning I suppose, that it (uric acid) is accumulating in the liver and spleen; on Day IV, salicylic acid is again taken, and in spite of rising acidity there is again a great rush of uric acid.

And there is a further point with regard to this figure; for on Day IV it appears that with the rush of uric acid there were some slight threatenings of headache in the morning. What does this mean? It will be seen on looking at the drugs taken on this day that I here made an abortive attempt to lessen the excretion of the uric acid under salicylate by taking alkali, and what happened was this, that during the alkaline tide part of the day (the morning) there was a rush of uric acid under the influence of alkali, and during the acid tide part of the day there was a rush of uric acid under salicylate. During the rush of uric acid under alkali the uric acid was present as uric acid in the blood, and caused slight headache; during the rush



Fig. 5.



Ac. Salicylic, 3ss.	} Well...
Sodae, gr. 40.	
Ac. Nitro-hydroc., 3ss.	
No drugs.	Well .....
No drugs.	Well .....
Sodae Salicylate, 3j.	} .....
" Bicarb., 3j.	
Some threatenings of headache in morning.	
Well .....	Well .....

under salicylate it was present in the blood as salicyluric acid and caused no headache. There was no headache on Day I under salicylate and acids, because the whole uric acid rush on this day took place after combination with salicylate, and the same applies to the days in Fig. 4, on which there was a large excretion of uric acid. Salicyluric acid is said to be present in the urine of persons taking salicylates; but uric acid is also present, as I have seen its crystals deposited from such urine on several

occasions; and it may be asked why is not the whole of the uric acid converted into salicyluric acid? The explanation is, I think, to be found in the two sources from which the uric acid excretion comes (see paper in 'Journal of Physiology'). I think that the uric acid which comes direct from the kidney without passing into the blood is not acted on by the salicylic acid and remains as uric acid in the urine, but that uric acid which has its source in the accumulation in the liver and spleen is acted on by the salicylic acid and converted into salicyluric acid. Once in the urine, both the uric and salicyluric acids are estimated as uric acid by Haycraft's process.<sup>1</sup>

As soon as I am able to obtain salicyluric acid in a state of chemical purity, I shall endeavour to test the question of its reaction with Haycraft's uric acid process, and also the questions connected with its solubility.

The facts, however, apart from these chemical explanations, have such an important bearing upon my previous work as well as on the action of salicylates in disease, that I do not wish to delay their publication for the indefinite period of time that may be necessary to obtain a final settlement of these chemical questions.

Some part of the explanation put forward must no doubt be regarded in the light of a working hypothesis to be replaced by a better whenever it may be necessary; but the one fact that salicylates can render the excretion of uric acid independent of acidity appears to me to afford an explanation of the action of these drugs in disease, the importance of which it would be difficult to exaggerate.

*Additional note on salicyluric acid, March 19th, 1888.*

I have said above that I thought it possible that the salicyluric acid, which is supposed to be formed by the combination of uric acid or of glycocine with salicylic acid, might on passing into the urine be estimated as uric acid by Haycraft's process.

<sup>1</sup> See additional note at end of paper.

As I was unable to obtain any salicyluric acid I endeavoured to test the question of its reaction with Haycraft's process by treating urine passed while taking salicylates, and I reached a point at which my residue ought, according to what is known of the chemistry of the process, to contain salicylic acid, salicyluric acid, and perhaps some traces of uric acid.

I then got some of this residue in solution and obtained a reaction with Haycraft's process, showing that about one sixteenth part of the residue consisted of uric acid.

Looking then to the fact that uric acid is quite insoluble in alcohol, while salicyluric acid is freely soluble in it, I treated the above residue with alcohol; and the alcoholic extract, which probably contained salicylic and salicyluric acid, gave no reaction whatever with Haycraft's process.

If these results are in any way to be depended on, salicyluric acid does not give any reaction with Haycraft's process, and when it is present in the urine cannot be estimated as uric acid; and the fact that salicyluric acid and its salts are generally more soluble than uric acid and its salts is in favour of this view, for if salicylurate of silver is soluble in ammonia it could not be estimated by Haycraft's process.

I am therefore obliged to suppose, in explanation of the results obtained with salicylates, that if the uric acid stored in the liver and spleen is converted into salicyluric acid in the blood, it is mostly reconverted into uric acid and salicylic acid as it passes the kidney, and only appears in the urine as salicyluric acid to a small extent.

I give these results for what they are worth because I see no immediate prospect of obtaining any pure salicyluric acid, and I have not been able to get any chemist to undertake its preparation.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 326.)

ON THE OCCURRENCE  
OF  
TUBERCULAR DISEASE OF THE TESTIS  
AS A LOCAL AFFECTION,  
PARTICULARLY WITH REFERENCE TO THE DESIRABILITY  
OF EARLY CASTRATION IN CERTAIN CASES.

BY  
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THE basis of the following paper is a report of five cases which came under observation in my out-patient practice, and which are recorded as they seem to present, for reasons afterwards discussed, some points of interest connected with the possible origin of tubercular testis as a purely local affection, its tendency to generalisation as shown by the implication of distant parts, especially the spine, the relation of the disintegration of the original disease to general tubercular infection, and the desirability of early castration in certain cases.

CASE 1. — In January, 1882, a boy, æt. 9, was brought to the hospital on account of a "swollen testicle." Four months previously he had received a

blow on the scrotum. Considerable swelling and great pain followed, but subsided almost entirely in a fortnight.

No further notice was taken of the matter till one month later, when the testicle became painful, swollen, and tender. The general health was not in any way affected and the boy attended school, running about much as usual, till three or four weeks before he came under my care, when rapid increase of the swelling occurred, and was followed, in a few days, by the bursting of an abscess over the affected organ. After this he became pale, thin and languid.

The family history was absolutely free from any evidence of tubercular disposition, the boy had always been well fed, healthily housed, and had, up to the date of the injury to the testicle, never suffered from a day's illness.

When first seen by me the patient was rather thin and pale. The right epididymis was much swollen, above it was nodulated, below the scrotum was boggy, and presented a sinus discharging thick pus. The testicle itself was somewhat hard. There was not much tenderness and very little pain. A most careful general examination failed to detect any sign of disease in other parts, including the opposite epididymis and testis, both seminal vesicles and ducts.

Three weeks later the condition of the testis was much the same; the boy was better in general health, but it was noticed that he held himself stiffly in walking. The spine was then found to be rigid and tender in the dorso-lumbar region. No pain in the back had been complained of and the mother was unaware of anything being wrong in that region. The seminal vesicles and left epididymis and testicle still showed no evidence of disease.

Although the mother was strongly urged to allow the boy to come into the hospital she declined to do so as she could not realise that the spinal disease was of consequence, and ceased to attend.

CASE 2. — A labourer, æt. 30, applied for treat-

ment in December, 1883. A year before he had contracted gonorrhœa, which was followed by "swelled testicle." When recovering from this he struck the testicle against the back of a chair, after which the pain and swelling recurred, then diminished, but never altogether disappeared.

Five months before coming under observation a small abscess burst at the lower part of the scrotum and remained continually open. His general health had in no way suffered. He thought nothing of the abscess, and only came to the hospital on account of a "touch of lumbago" which came on suddenly whilst he was lifting a heavy weight ten days before.

He had never suffered from syphilis, and the family history was perfect. In appearance the man was strong and healthy. The right testis was universally swollen, hard in some parts, boggy in others. At the lower part of the scrotum were two sinuses from which came curdy pus.

The spine at the junction of the dorsal and lumbar regions was perfectly rigid and tender on pressure. No disease could be detected in either seminal vesicle, duct, opposite epididymis, testicle, prostate gland, or other organ.

The patient could not be made to understand the serious character of his disease, declined to come into the hospital, and was not seen again.

CASE 3.—A cabman, æt. 30, applied at the hospital in January, 1884. Five months before, a blow on the left testicle was followed by much pain and swelling, which did not entirely subside. He had never suffered from gonorrhœa or syphilis, his family history was perfect, and he had always been a remarkably strong and healthy man.

On examination, the patient appeared to be in robust general health. The left epididymis was hard and nodular, there was some tenderness at the lower part, but no softening was apparent. The testicle itself seemed quite natural. A thorough investigation failed to find

disease in any other part, including the opposite testicle and epididymis, prostate gland, seminal vesicles, and ducts. After his first visit the patient was not seen again for six weeks, when he returned with more swelling about the testicle. At the lower part of the scrotum was a discharging sinus which had formed about ten days previously. The seminal vesicles, prostate, opposite epididymis, and testes remained healthy, but the dorso-lumbar spine was absolutely rigid, although he was entirely unaware of the existence of anything wrong with his back. Over the last dorsal spinous process, which seemed rather prominent, deep pressure elicited slight pain. As in the last case I could not induce the man to believe that the spine was seriously affected. He continued to attend for about two months, during which the disease steadily progressed, but the opposite testis, &c., did not become involved. He declined to consider the question of castration, and I ultimately lost sight of him.

CASE 4.—A messenger, æt. 16, first came under my care as an out-patient in May, 1884, on account of a swelling in the right side of the scrotum which had been caused by an injury received in the previous January. He had always enjoyed excellent health, had never suffered from gonorrhœa or syphilis, and his family history was perfect.

The lad was healthy looking and well nourished. The right epididymis was enlarged, nodular, slightly tender, but in no part soft. The testicle itself seemed normal, and no disease could be detected in the opposite testis, epididymis, seminal vesicles, or other organ. The testicle gradually became involved. At the end of June an abscess formed at the lower part of the epididymis and was laid open.

I then recommended castration, but the patient would not consent to the operation. A month subsequently some rigidity was detected in the spine over the lower dorsal region, deep pressure caused pain, but there was

no discomfort during ordinary movements. The seminal vesicles and opposite testis remained healthy. Castration was again proposed but negatived by the patient. A plaster spinal jacket was adapted and the boy went about his work as usual. A fortnight after this rapid swelling came on in the left epididymis, the testicle soon became affected, an abscess formed and was opened. The plaster jacket was not removed until September when the patient was taken into the hospital. The spinal disease had then increased considerably, the area of rigidity was greater, and there was commencing angular curvature. Some hard nodules could now be felt in the seminal vesicles and the vasa deferentia were much thickened. Both testicles were suppurating freely and discharging curdy pus through various sinuses.

A poroplastic support was now fitted, and he resumed his occupation, which he followed without discomfort till June, 1885, when he was readmitted on account of a fluctuating swelling in the right iliac region, from which a large quantity of unhealthy pus was removed by aspiration.

His general health now broke down, and after a number of complications, the most noteworthy of which was a typical attack of tubercular meningitis, from which he recovered, he ultimately died of general tuberculosis, as shown by post-mortem examination in June, 1886.

CASE 5.—A coachman, æt. 41, of strong and healthy appearance, commenced attending at the hospital in December, 1885, on account of some swelling about the right testis, which had followed directly upon an injury received about a year before. The swelling, which was extremely painful and tender at first, gradually diminished, but never entirely disappeared, subsequently it increased, and the pain and tenderness, which had quite left him, again came on.

He had always been remarkably well and strong, there was no suspicion of tubercular tendency in his family history, but he had suffered from a chancre twenty-three



years ago, which was followed by no other symptoms so far as he could recollect.

On examination, the right epididymis was large, hard, and nodular, the testicle itself was a little swollen and tender, but there was no sign of softening. No disease could be detected in any other part, inclusive of the opposite testicle and epididymis, both seminal vesicles and prostate.

At the beginning of January, some softening showed itself about the lower end of the epididymis. An incision was made and some thick pus evacuated. Castration was at this time urged upon the patient, but he declined the operation. Three weeks after this the spine was found to be quite rigid and very slightly tender on deep pressure over the dorso-lumbar region.

Again, the patient declined to allow castration to be performed. Some more breaking down about the epididymis occurred, and on March 23rd, 1886, he was admitted as an in-patient. At this date the spine showed extensive rigidity, there was pain on movement and much tenderness on manipulation. The scrotum was boggy on the affected side and the sinuses were discharging oily pus. There was no evidence of the invasion of any other parts by the disease. The general health had suffered curiously little.

By May 27th there was no indication of improvement in the condition of the spine in spite of absolute rest, the suppuration about the affected testicle and epididymis was more profuse, and the general condition of the patient was becoming unsatisfactory. Castration was now performed, the testicle after removal presenting an admirable example of tubercular disease, both to the naked eye and by the microscope.

Rapid improvement followed the operation, particularly in the condition of the spine, the rigidity decreased at a rate altogether out of proportion to the amount of rest enforced, and at the end of six weeks the patient considered the back perfectly well. The scrotal wound healed

soundly, but there was for two months some pain about the stump of the cord, which ultimately, however, settled down.

Four months after the operation all pain and tenderness had left the spine and there was merely a suspicion of rigidity. A leather spinal support was fitted and he was allowed to leave the Hospital.

When seen nearly a year after the operation, the spine was supple and entirely natural. He was following his occupation as a coachman. Not a sign of disease could be detected in any part of his body.

*Remarks.*—These cases were selected for record after a most thorough investigation, as they possessed certain important characteristics in common. In each instance the family history was perfect and there was an entire absence of privation, excess, or other condition predisposing in any way to the development of general tubercle. In every case there was, up to the time of the local affection, a perfectly clean bill of health, with the exception of patient No. 5, who had a chancre twenty-three years previously.

The original disease in all the cases was the result of direct irritation, traumatic in four, gonorrhœal in one. In every instance the spine became involved before there was any evidence of tubercle in the seminal vesicle, opposite testis or other part in the immediate neighbourhood of the original disease. The spinal affection was most insidious in its onset and was, at the time of its discovery by me, unsuspected by the patients, excepting No. 2, who found it out for himself accidentally.

In neither of the cases did the spine become implicated until after the breaking down of the original disease, nor did the general health become seriously affected until a considerable period after the formation of abscess.

The occurrence of tubercular disease in parts distant from the testis primarily attacked before the invasion of the corresponding seminal vesicle is, so far as I know,

not recognised as an ordinary or even occasional occurrence and may be taken, I presume, as fairly positive evidence of the commencement of general infection. Why in these particular cases the spinal column should be the part first to show signs of this secondary disease I do not attempt to explain.

The association of disease of the spine with tuberculous testis has been considered by more than one observer, particularly Reclus, who in his monograph on 'Tubercle of the Testicle,' devotes much attention to the matter, but there is, so far as I can ascertain, a remarkable paucity of clinical evidence as to which of the two parts is commonly the seat of the original disease. This is due mainly, I suppose, to the fact that as a rule patients do not come under observation until both parts have become implicated, and in some cases it is possible that the extremely insidious and painless onset of the spinal affection may have allowed it to escape the notice of the surgeon as well as of the patient. Be that as it may, there is in these cases, at all events, no doubt as to the testis being the seat of the original mischief.

The time at which the symptoms of the spinal implication first developed is a matter of much importance, for, as has already been pointed out, no sign of secondary disease of any part showed itself until the occurrence of disintegration and abscess in the part primarily affected, after which at no great interval infection surely followed. From this it is, I think, not unreasonable to infer that the risk of complication in other parts is greatest at the time of the breaking down of the original tubercular material, which therefore constitutes what I venture to call the "dangerous period," and may be taken, I believe, to apply not only to the disease as seen in the testicles but to any local tubercular deposit.

This close connection between the disintegration process referred to and the secondary manifestation of disease may, in the minds of some, no doubt admit of explanation by supposing that the two conditions are merely the outcome

of pre-existing general tubercle, but in these cases of mine there was no evidence whatever to excite even a suspicion of any tendency to general disease until after the breaking down of the part originally affected, in fact it is difficult to deny that they show as clearly as clinical observation can prove that the disease in the spine was the result of some infective action connected with the primary deposit in the testis.

The recognition of a tubercular disease, primarily of a purely local kind, which tends surely to the implication of other parts at a well-defined stage in the affection, must of necessity have an important bearing upon the treatment of such cases, as it is obvious that the general safety of the patient would be best provided for by the removal of the organ originally diseased upon the appearance of symptoms of disintegration indicating the onset of the "dangerous period."

It is, I believe, not unfrequently asserted that castration in the early stage of tubercular testis is unnecessary if not unjustifiable, since recovery may often occur without operative interference. If by this is meant merely that the abscesses in the testis may dry up and the sinuses heal, I admit the occurrence of such cases, but that recovery in the proper sense, *i. e.* without the implication of other parts, often occurs in those cases I venture to doubt, for I have examined a considerable number of patients bearing scars and other evidences of "cured" tubercular testis and cannot call to mind a single instance in which latent disease in some other part did not exist. Further, even if it be admitted that spontaneous recovery is possible, it cannot be denied that so long as tubercular material undergoing disintegration is present the patient is in constant danger of general infection; a fact in itself affording sufficient justification for the sacrifice of a diseased testicle, the loss of which, seeing that the organ is functionally impaired and possibly useless, can hardly be regarded as a serious calamity, excepting for sentimental or domestic

reasons, especially as the operation for its removal is without risk if done with ordinary care.

The rapid improvement in the condition of the spine in Case 5 after the extirpation of the suppurating testis is a remarkable instance of the advantage of castration after other parts have become affected, and is of interest in connection with the question of operation in the later stages of tuberculous disease.

A careful consideration of the cases recorded above from a clinical point of view appears to me to justify the following conclusions :

1. Inflammation of the testicle or epididymis as the consequence of injury or direct irritation may result in tuberculous disease of a purely local kind, which if left to itself tends surely to generalisation.

2. The greatest tendency to general infection is at a time subsequent to the breaking down of the original deposit, which may therefore be called the "dangerous period."

3. Parts remote from the testis primarily affected may be involved by the disease before it invades either seminal vesicle or opposite epididymis and testicle.

4. The rational treatment for these cases is castration upon the appearance of symptoms of suppuration about the original disease, *i. e.* at the onset of the "dangerous period."

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 329.)

A C A S E  
OF  
INTRA-PERITONEAL RUPTURE OF THE  
BLADDER;  
ABDOMINAL SECTION; SUTURE OF THE BLADDER;  
RECOVERY.

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C. H—, a healthy man, æt. 22, was admitted on the morning of March 1st, 1887, into Pitcairn Ward, complaining of pain in the lower part of the abdomen. He had spent the previous evening drinking in a public-house, and between the hours of eleven and twelve was leaving for the purpose of emptying his bladder which he felt to be uncomfortably full, when words arose with a companion and they went into the street "to fight it out." During the scuffle his antagonist butted him in the abdomen with his head and he fell to the ground. After a few minutes, however, he again continued the fight, notwithstanding that he was in great pain in the abdomen, and "finished" his assailant. He was then led home in a "doubled-up condition," passed the night in great agony, and was brought in a cab to the hospital about 10 o'clock on the following morning. He had been unable to pass any urine since the injury.

*On admission*, he was quite sober and rational and was

suffering from very little shock, indeed, he walked into the surgery with the assistance of two friends. Strangury was not present and there was only a dull pain above the pubes. On examination a fluid tumour was discovered in the lower part of the abdomen, but appeared to be too diffuse and to extend too high in the abdomen to be the bladder. The perineum was natural, and there was no history of stricture or other urinary trouble. A catheter (No. 6 silver) was found to pass easily but no urine flowed, although its point was felt to have entered the bladder by the finger in the rectum. It could not, however, be moved from side to side, the bladder appearing to be contracted around it; but on depressing the handle between the legs it was felt to free itself with a jerk, after which it moved more easily, and the point could be felt more plainly through the abdominal walls than natural. About six ounces of bloody fluid now escaped, and it was noticed that the fluid flowing through the catheter varied with the respiration. The temperature and pulse were normal, and there was no sign of peritonitis. Mr. Spencer, the house surgeon (now Assistant Surgeon to the Westminster Hospital), diagnosed an intra-peritoneal rupture of the bladder, and immediately sent for Mr. Walsham, who arrived at the hospital about 12 o'clock noon. The patient was at once taken into the operating theatre and placed under the influence of ether at 12.15 p.m., a little more than twelve hours after the injury. The skin of the abdomen having been thoroughly washed with soap and water, and afterwards well sponged with carbolic lotion (1 in 20), an exploratory incision about an inch and a half long was made in the middle line immediately above the pubes. The abdominal cavity below the reflexion of the peritoneum was then opened but no extravasated urine was discovered. The incision was next extended upwards for another inch and a half, and the peritoneum, the reflexion of which was felt to bulge, was cautiously opened by an incision about a quarter of an inch in length, when bloody fluid began to flow. On

enlarging the aperture in the peritoneum about four pints of the bloody urine and serum escaped, but the peritoneum did not appear in any way inflamed. On passing the fingers into the pelvis the point of the catheter was felt protruding through a rent in the bladder. The rent was vertical and entirely intra-peritoneal. It measured about an inch and a half, and its edges were fairly evenly divided. In the collapsed state of the bladder the rent appeared to commence immediately in front of the posterior reflexion of the peritoneum on to the lower fundus. Some little difficulty was experienced in obtaining a good view of the part in consequence of the contraction of the recti muscles, the bulging of the intestines into the wound, and the contracted state of the bladder, which lay deeply placed in the pelvis behind the pubes. To gain sufficient room the recti muscles had to be divided for about an inch transversely, but the parietal peritoneum was not included in the lateral cuts. The intestines being held aside the bladder was now hooked upwards and forwards into the wound and there retained by the fingers of an assistant whilst the rent was being sewn up. Nine Lembert's sutures of fine carbolized China silk were employed for this purpose. The sutures, which were inserted so as to ensure half an inch of the peritoneal surface of the bladder being in contact, were placed somewhat less than a quarter of an inch apart, and one suture was passed about a quarter of an inch beyond each end of the wound so as to prevent any leakage at the upper and lower angles. All the sutures were passed through the peritoneal and muscular coats only of the bladder, and were all placed *in situ* before any were tied. Previous to securing them the finger was introduced into the bladder and carried carefully along each side of the wound to make absolutely sure that none of the sutures had penetrated the mucous membrane. After the bladder had been sewn up a chromicized catgut suture was inserted between two of the silk sutures at a spot where the peritoneal surfaces did not appear to be quite satisfactorily in



contact. Eight ounces of a 1 per cent. boric acid solution were now forcibly injected into the bladder through a soft rubber catheter. The bladder became distended but there was absolutely no leakage. On removing the catheter the bladder contracted spontaneously and expelled the contained boric acid solution in a full and forcible stream through the urethra. The peritoneal cavity was then thoroughly irrigated with a 1 per cent. boric acid solution at a temperature of 98°. The irrigating can was held about five feet above the abdomen and the end of the india-rubber irrigating tube was passed freely amongst the intestines into all parts of the peritoneal cavity. The washing out was continued till the solution ran away quite clear, about two gallons of the fluid being used. Finally, the edges of the abdominal wound were brought together by China silk sutures passed through the skin and peritoneum and the superficial part of the wound was united by chromicized catgut, a carbolized, flat Gamgee's pad being placed over the intestines while the sutures were being introduced. The transverse cut in either rectus was not united by suture. The wound was freely sprinkled with iodoform and dressed with iodoform gauze and absorbent cotton wool secured by a domett bandage and strapping as after an ovariectomy. The carbolic spray was used throughout the operation, which lasted one hour and fifty-five minutes, including the time the patient was being brought under the anæsthetic and the preliminary cleansing of the skin of the abdomen. On the completion of the operation a soft rubber catheter was left in the bladder for about two hours, when it was removed and was only used on two occasions afterwards. The patient subsequently passed his urine spontaneously and continued to pass it every four hours, being reminded to do so by the nurse. There was comparatively little shock, and the pulse continued steady throughout the operation. The patient was ordered *Tinctura Opii*  $\text{m}\times$  every two or three hours while awake and to have no nourishment of any kind. He was allowed small pieces of ice to suck.

March 2nd.—Passed a fair night, but has not quite recovered from the shock of the operation. He has passed his urine every four hours both during the day and night, four to six ounces escaping on each occasion. It is acid, turbid, not clearing on boiling, and contains clots of mucus, a distinct cloud of albumen, and a little blood. The temperature (at 1.30) is  $101^{\circ}$ , the pulse 96, soft and slightly dicrotic. The abdomen is soft, moves freely during respiration, and is not distended. He has had no nourishment of any kind. Opium stopped.

3rd.—No sign of peritonitis; abdomen soft, moving with respiration, no pain. Pulse 96, soft, of good volume. Temperature (at 3.30)  $101.4^{\circ}$ . Thirty ounces of urine have passed during the last twenty-four hours (six ounces at 12 p.m.; eight ounces at 5 a.m., ten ounces at 9 a.m., eight ounces at 2 p.m.). The urine is still red in colour, and intimately mixed with blood. This morning ordered iced milk. Has had half a pint in four hours. Ordered two pints of iced milk in twenty-four hours and steam kettle for his cough, the result of some bronchitis set up by the ether.

4th.—Much brighter. No abdominal trouble whatever. Pulse 84. Temp.  $100.2^{\circ}$ . Passes his urine freely (forty ounces during the last twenty-four hours. 5 p.m., six ounces; 9 p.m., ten ounces; 12.35 a.m., four ounces; 5 a.m., twelve ounces; 10.30 a.m., nine ounces). The urine contains less blood, but is still distinctly red. Cough very troublesome. Spray from steam kettle continued. Linctus 3j occasionally.

5th.—No abdominal trouble. Pulse 96, soft. Temp. (at 10.15 a.m.)  $101.4^{\circ}$ ; (at 1.40 p.m.)  $102^{\circ}$ . Did not sleep well on account of his cough, which causes him pain in abdomen. Tinctura Opii  $\mathfrak{mxx}$  at 7.15 p.m., and again at 2 a.m. Urine thirty-nine ounces in twenty-four hours. It is lighter in colour but still smoky, and shows blood on guaiacum test; a slight cloud of albumen. Sp. gr. 1028. Takes two pints of milk in the twenty-four hours. Ordered beef-tea in addition to his milk.

6th.—At 7.45 p.m. yesterday (March 5th) the bowels were opened for the first time since the operation; a small ordinary motion passed. Slept well after one dose of *Tinctura Opii*. Passes urine every four hours. Urine contains very little blood. Temperature lower. Dressing changed for first time. Wound looking healthy. Re-sprinkled with iodoform, and dressed as before. To have an egg beaten up with his milk.

7th.—Doing well. Temperature averages 100°. Pulse 90. Tongue furred. Passed forty-four ounces of urine in twenty-four hours; four to eight ounces every four hours. Urine lighter in colour, and free from blood; acid; sp. gr. 1026, a distinct cloud of albumen. Has taken a pint of beef-tea, three pints of milk, and one egg. Ordered fish and soft bread. Wound dressed again. Given an enema of hot water. Small dark motion of normal consistency passed.

8th.—Excellent night, undisturbed by cough. Pulse 84. Temperature (at 8 a.m.) normal. Tongue clean. Wound again dressed. One stitch removed upper part. Slight suppuration over situation of divided recti; about one drachm of pus squeezed out. Passed forty-one ounces of urine in twenty-four hours. Urine lighter in colour; turbid; thick sediment at bottom of vessel; pus-corpuscles on microscopical examination.

9th.—Cough gone. Pulse 80. Urine slightly offensive on standing; acid; sp. gr. 1025; contains albumen and a few blood-corpuscles. Wound dressed, silk sutures all removed; deeper parts soundly healed. Some slight suppuration at seat of divided recti. Edges of wound supported by strips of strapping. Ordered two eggs and fifteen grains of chlorate of potash every four hours.

10th.—Temperature did not rise last night. Pulse 66. Passed forty-six ounces of urine in twenty-four hours. Less pus.

11th.—Passed forty-five ounces of urine in twenty-four hours. Contains less pus. No smell on standing.

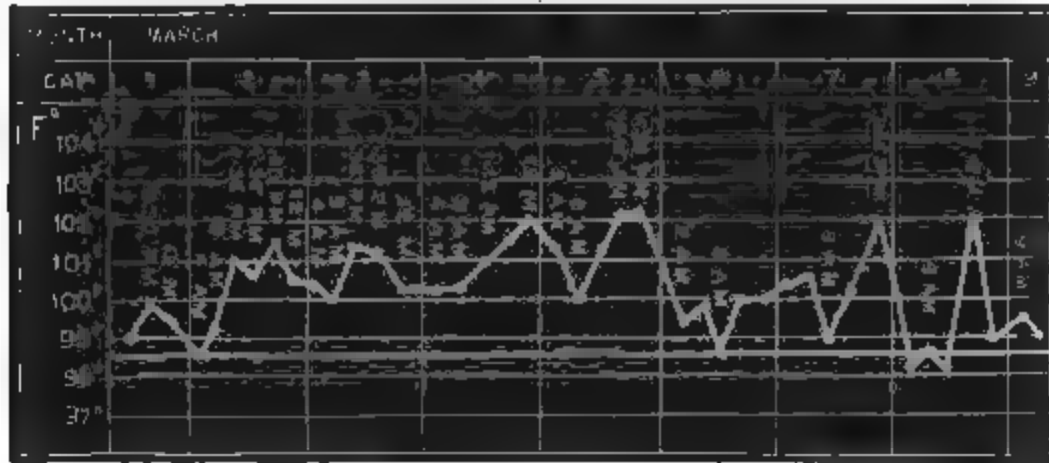
14th.—Placed on meat diet.

16th.—Urine free from albumen and pus. About fifty ounces passed daily.

24th.—Allowed to get up, wearing an abdominal belt.

April 1st.—Discharged.

*Remarks.*—There have now been, as far as I am able to ascertain, seventeen cases in which abdominal section has been performed for rupture of the bladder. The rupture in three of these cases was extra-peritoneal; in fourteen intra-peritoneal. Of the three cases of extra-peritoneal rupture two died and one recovered. They may be dismissed with the remark that in the case of the patient who recovered the wound was not sutured, but secured to the abdominal wall; whilst in the two



fatal cases death seems attributable to shock; in one, the anterior wall of the bladder was sutured and was found sound at the post-mortem examination; in the other, no rent in the bladder having been discovered on opening the abdomen, the abdominal wound was closed, and an incision made into the membranous urethra for the purpose of drainage; but at the autopsy the anterior wall of the bladder was found to have been perforated by a fragment of a fractured pelvis.

Of the fourteen intra-peritoneal ruptures (which more especially bear upon this case) the rent in the bladder was sutured in eleven cases; and in the remaining three a drainage-tube was placed in the wound.

To dismiss first the three cases in which no sutures were employed, one recovered and two died, death being

due in one of the fatal cases to peritonitis, and in the other to suppression of urine, the result, probably, of kidney mischief brought on by long-standing stricture of the urethra.

Lastly, of the eleven cases in which the rent in the bladder was secured by suture, five recovered and six died. Of the six fatal cases, death in three was due to peritonitis; in two it was probably the result of shock, and in one of hæmorrhage from a wound in the perineum, which had been previously made for the purpose of exploring the condition of the bladder. In the three cases in which death was the result of peritonitis, in one the suture (in this case a continuous catgut) had given way, and in the other two there was a leakage at the lower part of the wound. In two of these cases, moreover, the operation was not performed till twenty-nine and forty-eight hours respectively after the injury, the sutures were passed through all the coats of the bladder, the peritoneal cavity was not completely sponged out, and a drain-tube was employed. Further, in one of them peritonitis had already set in at the time of the operation. In the two cases in which death occurred from shock, and in the case in which it was caused by hæmorrhage, the bladder was found watertight in one (Case 4) and there was no peritonitis, but it leaked at the lower angle of the wound in another (Case 9). One of these, moreover (Case 9), was complicated by a fractured pelvis. Turning to the five successful cases in which suture was employed, the operation in all was performed within twenty-seven hours of the injury; in none had peritonitis supervened. In all the sutures were passed through the muscular and peritoneal coats only, and the peritoneal cavity was thoroughly washed out. In one of them only was a drain-tube employed. In three a catheter was retained in the bladder, though in one of these it was removed two hours afterwards. In one (Case 14) the rent was partially extra-peritoneal, and the extra-peritoneal wound was left open to serve as a drain.

Although of the eleven cases in which the rent in the

bladder was sutured more than half died, the result, when the cases are critically examined, is very encouraging; and points, I think, to this being the method which, as a matter of routine, will in the future be commonly employed in uncomplicated cases. The lessons, I think, that these cases teach are:

1stly. The desirability of early resort to operative measures, as although it would appear that the urine may remain for many hours in the peritoneal cavity without necessarily setting up peritonitis, in all the successful cases the operation was undertaken within twenty-seven hours of the injury. The importance, therefore, of an early diagnosis cannot be over-rated. The points chiefly relied on for diagnosis in the present case were:—(A) The history that the bladder was full at the time of injury; (B) the non-escape of urine notwithstanding that the catheter was ascertained to be in the bladder by the finger in the rectum; (C) the subsequent escape of bloody fluid after the point of the catheter had been felt to free itself with a jerk on depressing the handle; (D) the fact that the point could be then felt through the abdominal walls more readily than under normal circumstances; and (E) the flow of bloody urine varying with the respiration. The case was so clearly one of ruptured bladder that I had no hesitation in at once opening the abdomen. But in a doubtful case I should certainly before doing so inject the bladder through a catheter with ten or twelve ounces of some antiseptic solution, and then note if the same quantity could be subsequently drawn off.

2ndly. These cases show the importance of employing a suture that will not become softened and give way, and of making sure by forcible injection of the bladder that the viscus has been rendered thoroughly watertight. In four of the unsuccessful cases the fatal result may, I think, undoubtedly be attributed to the leakage through the wound at one spot. It is therefore most necessary that attention should be paid to this point, and especially to the securing of the lower and posterior part of the

wound, as it was here in three instances that the leakage occurred. This danger of leakage may perhaps best be avoided by following the suggestion of Sir William Mac Cormac, of placing at least one suture beyond the angles of the wound.

3rdly. With regard to the cleansing of the peritoneal cavity, irrigation would appear to be preferable to sponging out; but so long as the cleansing is thoroughly done and the fluid is of a non-irritating character it does not seem to matter what fluid is used. In four cases, boric acid (1 per cent.) was employed; in one, sublimate solution; in another merely warm water.

4thly. A preliminary incision in the perineum for the purpose of exploring the bladder, or subsequently with the object of drainage, does not commend itself for adoption. The exploration in doubtful cases can be better performed through an incision above the pubes, not necessarily opening the peritoneal cavity till after the upper fundus of the bladder has been examined and found sound; whilst a subsequent incision in the perineum can only do harm by allowing septic changes to occur and spread to the bladder. In Mr. Teale's case the fatal result was due to hæmorrhage from the perineal incision.

Lastly. The tying in of a catheter after the operation appears to be quite unnecessary if the rent in the bladder is securely sutured and care is taken that the patient passes urine every four or five hours. The presence of a catheter, moreover, as in Hofmohl's case, seems attended with the risk of cystitis, and subsequently of septic changes occurring in the bladder wound. In my own case the catheter was removed after two hours; in Sir William Mac Cormac's second case and in Mr. Holmes's case it was not used at all.

A table of the seventeen cases in which abdominal section has been employed for ruptured bladder is here added. Sixteen of these appear in Sir William Mac Cormac's table appended to his interesting paper on abdominal section.





*Seventeen Cases of Ruptured Bladder treated by Abdominal Section.*

## A. INTRA-PERITONEAL.

Case	Reference.	Surgeon.	Age	Cause.	Date after injury.	Condition of peritoneum.	Size and condition of rent in bladder.	Kind of suture.	Treatment of peritoneum.	Incision in perineum.	Catheter in bladder.	After-treatment.	Remarks.	Death or recovery.
1	St. Barth. Hosp. Rep., 1876	Willet	48	Kick	29 hours	Peritonitis	3 1/4 inches, oblique, superior and posterior, ragged below	8 China silk, 1/4 inch apart, through all coats	Partially washed out, drain-tube	No	Yes	—	Peritonitis; lower angle of wound given way	Death.
2	Med.-Chir. Trans., vol. lxii, p. 335	Heath	47	Blow	42 1/2 hours	Much blood present	2 inches, vertical, posterior	Continuous catgut, through all coats	Partially sponged out, drain-tube	No	Yes	—	Peritonitis; lower angle of wound given way	Death.
3	Lancet, 1886, vol. ii, p. 972	McGill	34	Contact iron gate	66 hours	Contained a pint of urine; no peritonitis	4 inches, superior and posterior	9 chromic gut; mucous membrane not included	Washed out with 2 galls. boric acid solution; peritoneum closed; incision in bladder in front of peritoneum for drain-tube; spray	No	—	—	Died 18 hours afterwards, probably of shock; no peritonitis	Death.
4	Lancet, 1887, vol. i, p. 1133	Teale	25	Kick	—	Large quantity straw-	1 inch, posterior	6 fine catgut, Lambert's	Closed; not stated if washed	Yes, for exploration	No	—	Died 7 hours after of hæmorrhage	Death.

5	Lancet, 1886, vol. ii, p. 1118	Mac Cormac	33	Ran against post	19 hours	fluid; no peritonitis	4 inches, median, vertical, posterior, superior	16 Lembert sutures, first and last beyond limit of wound; catgut between; no leakage	Irrigated 2 galls. boric acid at 98° till fluid clear; no sponging; spray; drain-tube 4 days	No	Yes	Catheter removed 3rd day; not again needed	No bad symptom	Recovery.
6	Lancet, 1886, vol. ii, p. 1119	Mac Cormac	37	Fell in sitting posture	27 hours	Large quantity of urine and serum; no peritonitis	2 inches, oblique, superior and posterior, left side	12 fine silk Lembert's, one above and below rent; no leakage	Irrigated, boric acid, 98°; no drain-tube	No	No	—	External wound united in a week	Recovery.
7	Unpublished	Walsham	22	Blow	12 hours	4 pints blood-stained fluid; no peritonitis	1½ inches, vertical, posterior, fairly even	9 China silk Lembert's sutures, one above and one below rent; no leakage	Irrigated boric acid, 1 p.c., 98°, till ran clear; no drain	No	Yes, 2 hours only	No nourishment for 24 hours; opium every 2 hours; sutures removed	No bad symptom	Recovery.
8	Lancet, 1887, vol. ii, p. 153	Holmes	24	Kick	6 hours	—	2 inches	8 fine silk; peritoneal and muscular coats only	No spray; washed out with warm water	Yes, median; soft tube in urethra, not in bladder	No	—	Did not drain through tube	Recovery.

Case	Reference.	Surgeon.	Age	Cause.	Date after injury.	Condition of peritoneum.	Size and condition of rent in bladder.	Kind of suture.	Treatment of peritoneum.	Incision in perineum.	Catheter in bladder.	After-treatment.	Remarks.	Death or recovery.
9	Annals of Surgery, 1885, vol. i, p. 67	Bull	46	Fall, 16 feet	13 hours	Bloody urine	3½ inches, posterior	7 Lembert's carbolized silk; not tested for leakage	Sponged out; intestines were drawn out of wound and protected by warm moist towels	No	Yes	—	Died 7 hours after; fractured pelvis; greatshock, comatose; wound leaked, lower angle	Death.
10	Lancet, 1886, vol. ii, p. 399	Duncan	38	Passage of wheel	22 hours	Bloody urine; peritonitis	2½ inches, posterior	No sutures	Washed out; glass tube in Douglas's pouch	Yes, tube brought through incision	No	Nutrient enemata; small doses of sulphate of magnesia	Died 79 hours after; suppression of urine; kidney mischief result of stricture	Death.
11	Centralblatt für Chirurgie, 1885, p. 838	Sonnenberg	—	Fall	48 hours	Peritonitis	Vertex to neck, posterior	No sutures	Drain-tube, through upper part of abdominal wound	No	No	—	Died on 4th day; intense peritonitis; urinary infiltration in pelvis	Death.
12	Unpublished, MacCormac's Abdominal Section, p. 54	Walters, of Pittsburgh	22	—	10 hours	Bloody urine; signs of peritonitis	Extensive rent at base	No sutures	Drain-tube; clots sponged out	No	Yes	Not stated	—	Recovery.
13	Unpublished, MacCormac's	Symonds	7,	—	7 hours	Bloody urine	V-shaped; partly	12 Lembert	Sponged out	No	No	Peritonium reopened	Died in 7 days; bladder	Death.

14	Unpub- lished, Mac Cormac's Abdominal Section, p. 52	Hofmohl	27	Fell from win- dow, frac- ture of pelvis	10 hours	Much bloody fluid	1 1/2 centi- metres; also extra-peri- toneal rent behind pubes	2 rows carbolized silk; edges pared where irregular	Sponged out with sublimite tampons; extra-peri- toneal rent only partially closed to act as drain	No	Yes	Opium, ice over abdomen	peritonitis Cystitis; urethritis	Re- covery.
B. EXTRA-PERITONEAL.														
15	Unpub- lished, Mac Cormac's Abdominal Section, p. 52	Fox	38	—	19 hours	Bloody serum	2 1/2 inches, anterior wall, extra- perito- neal	15 Lembert	Not stated	Not stated	Yes	Not stated	Died in 42 hours; bladder wound firm; great shock	Death.
16	Unpub- lished, Mac Cormac, Abdominal Section, p. 53	Mayo Robson	68	—	3 hours	Not injured	Anterior wall; frag- ment of frac- tured pelvis in rent; ex- tra-peri- toneal	Laparo- tomy; no rent found	Wound closed	Incision in mem- braneous urethra, and tube passed into bladder	No	Not stated	Died in a few hours from shock	Death.
17	Annals of Surgery, Feb., 1887	Socin and Keser	20	—	—	Not injured	Anterior wall; extra- perito- neal (?)	Laparo- tomy; bladder wound sutured to abdominal wall	Drain-tube 9 days	No	No	—	—	Re- covery.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 333.)

# RELAPSING TYPHLITIS TREATED BY OPERATION.

BY

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TYPHLITIS, perityphlitis, and paratyphlitis are terms that, in the light of recent pathology, are losing no little of their original meaning. The distinctions that separated them have in great part vanished. The terms are now practically synonymous, and refer mainly to an inflammation in the vicinity of the cæcum. This inflammation will be chiefly peritoneal, and need not involve the cæcum itself either primarily or secondarily. Paratyphlitis, or inflammation of the subserous connective tissue in the cæcal region, is ceasing to be a disease with a clear individuality. It possibly belongs to the time when it was believed that the cæcum possessed a non-peritoneal surface. Since it has been shown that that bowel is always entirely enveloped by peritoneum the condition has been less heard of.

The so-called subserous abscess, the result of paratyphlitis, proves in the majority of cases to be an encysted peritonitis. There is no more reason why the subserous connective tissue in the cæcal district should inflame

under supposed irritation than would apply to the tissue outside a hernial sac.

The term typhlitis, as implying an inflammation of the cæcum, is usually a misnomer. It is the vermiform appendix and not the caput coli that plays the most important part in these grave affections.

In the greater number of fatal cases of typhlitis the cæcum has been found intact, while the appendix is inflamed and probably perforated. The perityphlitic abscess is usually—possibly always—an encysted peritonitis, and the cause of that peritonitis is to be found more often in the appendix than in the cæcum.

Typhlitis, or inflammation of the cæcum, is not to be denied. Ulcers—and notably stercoral ulcers, due to the impaction of fæces in the bowel—are not uncommon in the cæcum. These may lead to perforation, to perityphlitis, and possibly to inflammation of the connective tissue that is not far distant.

The overwhelming influence of the appendix has been well demonstrated by Fitz<sup>1</sup>, With<sup>2</sup>, Matterstock<sup>3</sup>, and others.

The appendix is a part of the human body that is in reality obsolete, and exists as an organic anachronism. It is the deformed relict of the elongated cæcum of lower mammals—of the capacious cæcum of the rodents, and the remarkable blind gut of the marsupials. It would appear to be functionless and to be perishing (as do many obsolete organs) by a pathological process. So high is the mortality in typhlitis, and so common is the disease, that the appendix may be conceived to take some share in evolution, since in the question of the survival of the fittest an advantage would attach to the being whose appendix was abortive or had been obliterated by early disease.

It is significant in this relationship that the tendencies

<sup>1</sup> 'International Journal of the Medical Sciences,' Oct., 1886, p. 321.

<sup>2</sup> 'London Medical Record (Abstract), 1880, viii, p. 213.

<sup>3</sup> Gerhardt's 'Handb. d. Kinderkrankh.,' 1880, iv, 2, p. 897.

of diseases of the appendix are to lead to obliteration of that process, and pathology makes it clear that when once the process is involved no better conclusion can follow than that marked by the practical disappearance of this part of the intestine. Indeed, if it be true that a sure guide in active treatment is to imitate the results of such undisturbed morbid changes as lead to cure, then the extirpation of the appendix would appear to be suggested in a somewhat wide extending phase of disease.

The frequency with which the appendix proves on post-mortem examination to have been the seat of disturbance is quite remarkable. Tungal<sup>1</sup> states that during two years he found in the post-mortem rooms of the Hamburg Hospital no less than ninety-six examples of gross disease of the vermiform process. Toft<sup>2</sup> goes further, and has endeavoured to show that one individual in three possesses a diseased appendix. My own investigations, I must confess, do not lead me to place this proportion quite so high.

In my Hunterian Lectures delivered at the College of Surgeons in 1885 I gave an account of the condition of the appendix derived from the examination of 100 fresh bodies. The growth of the process is uncertain. It appears to attain its full dimensions very early in life. It fluctuates in length from one inch to six inches or more. Its variations in outline are even more extreme. Owing to a certain condition of the mesentery of this process it is apt to become contorted and bent. This bending of the tube may lead to its obliteration, while inflammation may be excited by retention of mucus and other matters in the distal end of the canal. It is common to find the free end of the appendix greatly enlarged and distended from this cause. Moreover, the tube when acutely bent upon itself may be found ulcerated at the seat of flexion, and it is easy to understand that that ulceration may lead to a more widely-spread inflammation. In the majority of cases the vermiform process lies behind the end of the

<sup>1</sup> Matterstock, loc. cit.

<sup>2</sup> With, loc. cit.



ileum, pointing towards the spleen, but from disease or from simple anatomical changes in its mesentery it may be found lying in all possible positions. Its posture with reference to the general cavity of the abdomen is, of course, mainly dependent upon the mobility of the cæcum. With remarkable frequency the appendix is found to have contracted adhesions to neighbouring points of the peritoneum, a condition that may lead to other and more serious troubles than typhlitis.

These are some of the anatomical conditions in the appendix that may lead to typhlitis, but a still commoner cause of the trouble remains. The muscular coat of this little tube is very feebly developed, and this circumstance, combined with its position and its arrangement as a cul-de-sac, greatly favours the accumulation of matters in its lumen. These take the form occasionally of foreign bodies derived from the food, but more frequently consist of inspissated fæces. Fitz,<sup>1</sup> in his able monograph on the appendix, states that more than three fifths of all cases of perforated appendices are due to concretions and foreign substances lodged in the little canal.

When the appendix does become inflamed, anatomical conditions assist in rendering that inflammation local. In 18 per cent. of all cases I found the process lodged behind the cæcum; and, when in this posture, a perforation would have every prospect of leading to a mere local peritonitis. The extent of fixed peritoneum in the vicinity of the appendix, and the arrangement of the bowel about the cæcum, all encourage the formation of adhesions and assist in localising inflammatory exudations.

A study of the appendix and of the part it plays in the production of typhlitis shows this—that if by some magic an attack of typhlitis could be foretold, it could be prevented in a large proportion of cases by a removal of the appendix itself, or at least by some operation that would remedy the lesions or defects of which it was the seat.

It must not be assumed that every inflammatory dis-

<sup>1</sup> 'International Journal of the Medical Sciences,' Oct., 1886, p. 328.

turbance of the appendix will lead to phenomena that could be classed with typhlitis. Fitz observes that attacks of inflammation frequently occur in the vermiform process without giving rise to any characteristic symptoms, and often without a suggestion of any distinct malady. I met, in the post-mortem room, with three cases of obliteration of the appendix by inflammation in patients whose medical histories, as written in the ward notes, showed that they had never had notable abdominal trouble.

There are cases, however, where operative interference is distinctly indicated in typhlitis, not so much for the purpose of dealing with existing symptoms, as of warding off further troubles.

When peritonitis has been induced, or when a perityphilitic abscess exists or a localised collection in the serous membrane is recognised, the treatment is plain enough; the abdomen is opened and the inflammatory exudation is treated as are like collections elsewhere, viz. by free incision and drainage.

These, however, are not the only cases that appear to me to demand operation. Typhlitis is very liable to relapse. Fitz has shown that successive attacks occur in 11 per cent. of the cases. The attacks may be numerous. Eichhorst cites the case of a man who had five attacks in one and a half years. I am under the impression that the great majority of examples of relapsing typhlitis are due to troubles in the appendix. Indeed, that diverticulum affords anatomical reasons for relapse that are not provided either by the cæcum or by the peritoneum. A simple local peritonitis shows no disposition to relapse, nor are there circumstances in those lesions of the cæcum that lead to typhlitis, which will readily explain frequent attacks of inflammatory trouble.

If these premises are well founded, relapsing typhilitis should be readily treated by operation in a certain proportion—and probably in a very large proportion—of cases.

The following case serves to illustrate the matter, and has afforded the only opportunity I have had of carrying

this proposed treatment into practice. The case was simple and the patient made a sound recovery.

A man, æt. 34, a working engineer, was admitted into the London Hospital under the care of Dr. Stephen Mackenzie, on December 31st, 1886, suffering from typhlitis. He had been a healthy, vigorous man of sober habits. He had had no illness except an attack of bronchitis fifteen years ago. He had been free from abdominal trouble previous to the present affection and his bowels had always been regular. Early in August, 1886, he was seized with pain in the right iliac fossa, followed by local swelling, vomiting, and constipation. This was his first attack of typhlitis. He was confined to bed and for a while improved under treatment, but after a week or so of freedom from pain, the symptoms reappeared with renewed vigour, the swelling and tenderness in the iliac fossa became once more pronounced, and the patient, to use his own expression, "was as bad as ever again." Between the attacks he was not well enough to leave his bed, nor was he ever quite free from a sense of discomfort in the cæcal region. He was confined to bed for three months, *i. e.* from the commencement of August to the end of October. In November he was able to resume his work, and for a while complained of no trouble in his abdomen. He found, however, that his bowels did not act so freely after his illness as before. He remained well until December 27th—four days before his admission into the hospital—when he was once more seized with pain in the right iliac region attended by local swelling, sickness, and much prostration. He at once took to bed, but as his symptoms did not abate he came to the hospital.

When admitted he presented the ordinary symptoms of typhilitis. He was well nourished and exhibited no organic disease other than that in the abdomen. A globular swelling occupied the right iliac fossa and was the seat of extreme tenderness. The abdomen was but slightly distended, the bowels were confined, but there was no

vomiting. His temperature was  $99.2^{\circ}$ . Opium was given and the constipation was relieved by enemata. His progress was very slow, the pain in the abdomen persisted, the swelling remained tender and diminished but sluggishly. It was not until February 8th, some six weeks after the commencement of the attack, that the abdominal symptoms had entirely disappeared. The patient was much weakened by his confinement in bed, but he complained of no pain or tenderness in his abdomen, and no trace of any iliac swelling was left. Dr. Mackenzie now requested me to see the patient with reference to possible operative interference. Dr. Mackenzie was of opinion that the patient would be the subject of further attacks, and endorsed the suggestion that the abdomen should be opened and the cæcal region explored in the hope that the cause of the trouble might be removed.

I performed laparotomy on February 16th. I opened the abdomen by a vertical incision immediately over the region of the cæcum. That part of the bowel was soon exposed. It was free from adhesions. There were evidences of old peritonitis about the appendix. To the base of that process the omentum had become adherent. The mesentery of the appendix was shrunken and had bent the tube upon itself at an acute angle. The part of the process on the proximal side of the bend was of normal aspect, that on the distal side was distended, was greatly thickened, and formed a rounded swelling the size of the end of the forefinger. I had intended to remove the appendix, but it at once became straight on cutting away the omentum. I bared the convex side of the tube of peritoneum, thinking that fresh adhesions might occur upon that side and so hold the appendix in a straight position. The whole process only measured two inches and a half. I set free the omentum, sutured a small rent in the peritoneum that had been made during the examination, and closed the abdominal wound. It was evident that the shrinking of the appendix mesentery had so bent that tube as to occlude it. Mucus had

accummulated beyond the bend, and becoming retained had excited the peritonitis. The lumen had not become obliterated and the end of the diverticulum had apparently not been perforated.

The patient recovered without a bad symptom. On March 18th, some little time after he was up and about, he had some slight pain in the seat of the old trouble. It was apparently due to constipation, as it disappeared at once on the administration of an aperient. The patient was kept in the hospital for another month, in order to be under observation. He remained perfectly well and has since that time had no further abdominal trouble.

In the majority of cases it would probably be wiser to remove the appendix. If this is done as much care must be taken to close the divided end of the tube as would be taken to close a hole in the small intestine. A mere ligature would not be safe. Two sutures would suffice to bring the mucous membrane together, and the peritoneum should then be adjusted over this line of union by several points of Lembert's suture.

It must be remembered that peritoneal adhesions or a peritoneum scarred by adhesions will not possess the same plastic power as the normal membrane.

In the event of the process being so greatly thickened that suture of its serous coat is impossible I should be disposed to fix the stump to the nearest point on the surrounding parietal peritoneum.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 337.)

**A CASE**  
**OF**  
**NEGLECTED DISLOCATION OF THE**  
**HUMERUS**  
**FOLLOWED BY**  
**PARALYSIS OF THE NERVES OF THE**  
**HAND AND FOREARM,**  
**TREATED BY EXCISION OF THE HEAD OF THE HUMERUS.**

**BY**  
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A HEALTHY man, æt. 48, a waiter by occupation, was admitted into Charing Cross Hospital on September 3rd, 1887. Eleven weeks previously he had sustained an injury to the left shoulder, the true nature of which seems not to have been suspected. Severe pain and œdema of the limb followed the accident, and on the subsidence of this swelling the hand was noticed to be getting numb and useless, and these latter symptoms have steadily increased. The previous history of the patient was in all respects satisfactory. On examination of the affected limb the ordinary evidences of “subcoracoid” dislocation were present. There was slight wasting of

the deltoid muscle and also of the flexor muscles along the ulnar side of the forearm. All the signs of ulnar nerve paralysis were marked. The interosseous muscles had greatly wasted, so that the shafts of the metacarpal bones were unduly prominent. Sensation had totally disappeared in the skin supplied by this nerve. The median nerve was also implicated, but not to so marked a degree. Sensation was greatly impaired over the skin of the front of the index and middle fingers, and the short muscles had wasted somewhat. The skin of the hand and fingers was congested, of a glossy appearance, smooth and cold to the touch. The nails were curved over the ends of the semi-flexed fingers. The radial pulse was much diminished in force on the affected side.

The patient expressed himself to the effect that the hand was quite useless to him, and he was ready to undergo any treatment that promised relief. About twelve weeks after the accident he was placed under an anæsthetic, and I attempted to reduce the dislocation by the ordinary methods of manipulation, with extension and counter-extension. The pulleys were not employed. It was quite obvious that unless a great deal of force were employed the head of the bone would not move from its fixed position, and that even if forcible and prolonged attempts at reduction were resorted to the success attending them would be dubious. I therefore proceeded to excise the head of the humerus. This was accomplished by means of the anterior longitudinal incision usually adopted. The head of the bone lay at a great depth. Owing to this circumstance, as well as the presence of numerous fibrous adhesions, the operation was attended with some difficulty. Bearing in mind the probable close relation of some of the more important axillary structures to the displaced bone, I peeled the parts from the latter with a raspatory, only using the knife to divide structures of a more resistant nature *e. g.* the muscles attached to the tuberosities. In this way the head of the bone was finally cleaned, protruded from

the wound, and sawn off through the anatomical neck. The medullary canal was not opened, and the broad surface left was levelled at its margins with cutting forceps so as to leave a rounded end to the bone. The circumflex vessel bled very freely, and needed ligature. A large drainage-tube was introduced, a pad fixed in the axilla, and carbolised gauze dressings employed. The case progressed uninterruptedly towards recovery, and I need not occupy the time of the Society with details of dressings or symptoms. The radial pulse was restored to its ordinary strength immediately after the operation. The third day after the operation the condition of the hand was carefully tested, and it was noted that sensation had returned in all the parts supplied by the median and ulnar nerves, except the dorsal aspect of the little finger, which was quite numb and remained so for several weeks. The patient left the hospital with the wound soundly healed twenty-three days after the performance of the operation. Already improvement was manifest. He could pick up and hold light objects. Sensation had returned to all parts except the back of the little finger; here it was imperfect. He was ordered to still wear the axillary pad, and to have the affected parts galvanised thrice weekly.

On the 1st of November massage was commenced to the affected limb, and carried out about three times a week.

Twelve weeks after the operation, being the same time that had elapsed between the accident and the operation, I examined this patient, and this is a summary of his condition. The interosseous muscles have partly regained their form and plumpness. The movements of the thumb are strong and good. The muscles of the little finger are still weak and flabby. The man, however, declares that the strength of this digit is improving. The movements at the shoulder are, on the whole, satisfactory. He can use his hand and arm freely in the exercise of his vocation, being able to hold plates, and perform the duties of a waiter at a large London hotel. Further im-



provement of the condition of the muscles of the little finger may fairly be looked for.

I have ventured to bring a brief account of this case before the Society, because I think it suggests a useful mode of treatment for those dislocations of the shoulder which are associated with symptoms of pressure on the large nerve-trunks, for although it is true that in some of these cases restoration of function of the nerves ensues, yet this result is altogether uncertain, and in a considerable proportion the wasting of the muscles and inutility of the limb remain permanent. It is generally allowed that the process of reduction of old dislocations of the shoulder is attended with considerable risk. It is difficult, if not impossible, to estimate what degree of force is to be employed which will suffice to overcome the resistance offered by adhesions and structurally shortened muscles, and yet leave uninjured vessels and nerves which may be incorporated with them by the process of inflammation. Often also after a degree of force has been employed which only the necessity of the case could demand, the displacement remains, and risks have been encountered for no very useful result. In a comparatively short surgical experience I could relate several disasters following attempts at the reduction of old dislocations of the humerus, under the hands of surgeons whose names are sufficient guarantee that no needless force was employed. It is questionable whether in old dislocations as useful an arm cannot be obtained at a minimum of risk, by passive motion sedulously applied, and massage of the affected muscles. Should excision be deemed needful it is fair to state that though dangers may attend the actual performance of the operation, its after-risks are not more considerable than when the operation is performed for compound injuries or disease, and if due caution be exercised in the performance of the operation it seems in my judgment a less risky and more advisable expedient to adopt than the traction of pulleys, or the efforts of relays of assistants. In the present instance, though

perfect success has not been obtained, such marked improvement resulted from the operation that the performance of it may with justice be taken into consideration in a similar case. When nerve-trunks have been contused or stretched a considerable time must elapse before their functions are fully restored. Hence we may fairly hope for still further improvement in the muscular power of the hand and forearm in this case.

The literature of what may be termed the operative treatment of ancient dislocations of the shoulder is, so far as I have been able to ascertain, not very extensive. Subcutaneous division of the capsule of the joint, and of resisting bands of adhesions, is highly spoken of by Andrews.<sup>1</sup> Mr. Adams published a letter in the 'British Medical Journal,' January 20th, 1877, from Mears, of Philadelphia, relating the application of Adams' operation of subcutaneous osteotomy to the surgical neck of the humerus in a case of unreduced subcoracoid dislocation of over two years' standing. The pain was relieved, and according to the testimony of Pancoast a surprising degree of motion was restored, but no mention is made of the presence of nerve-lesions in this case. Division of the muscles and tendons has also been adopted.

Warren<sup>2</sup> excised the head of the humerus in 1860, on account of an old dislocation. A V-shaped incision was made through the soft parts, and the bone divided below the tuberosities, the head being afterwards dissected out. Great and permanent relief followed from the symptoms of nerve-pressure before present.

Hamilton<sup>3</sup> states that Desprès designedly fractured the neck of the humerus upon two occasions, but the cases are not commented upon favorably; and he refers to operations of this nature by Volkmann, Cramer and Kuster.

Volkmann seems to have practised the operation in two

<sup>1</sup> Ashhurst, 'Encyclopædia of Surgery,' 1882, vol. iii, pp. 671, 672.

<sup>2</sup> 'American Journal of Medical Science,' April, 1876, p. 452.

<sup>3</sup> Hamilton, 'Fractures and Dislocations,' 7th Amer. ed., Philad., 1884, p. 711.

instances. His first case was that of a man, aged fifty-three. The dislocation was of the "subcoracoid" variety and had existed for five weeks. Ordinary means of reduction had failed. The incision was made through the axillary space. Free bleeding occurred from the axillary vein, which was found perforated by a spicule of bone. The vein was tied, but there seems to have been great difficulty in getting the bone into good position. Some improvement, however, resulted.

The second case of excision was performed for repeated spontaneous dislocation. The incision was anterior, and great improvement resulted from the operation.

Professor Annandale<sup>1</sup> performed an operation of this kind, and it is the only one I have been able to find recorded in the surgical literature of this country. A woman aged sixty-two had suffered from an unreduced dislocation of the humerus under the clavicle for six weeks. There was severe pain from pressure on the nerves. The incision, which exposed the head of the bone, divided the circumflex artery so close to the main trunk that the operator was obliged to ligate the latter above and below the aperture. Gangrene of the arm ensued, amputation was necessitated and death resulted.

Excision of the head of the humerus for old dislocation is alluded to in the last edition of 'Erichsen's Surgery' edited by Beck.<sup>2</sup> Holmes also alludes to the possibility of its performance in the second edition of his work.<sup>3</sup> Billroth<sup>4</sup> recommends excision of the head of the bone when pressure on the large nerves is marked, and reduction has been found impossible. He states that he has seen a case of the kind, and considerable improvement followed the operation, though paralysis was not completely removed. Gross<sup>5</sup> also alludes to Warren's case, and views the operation with approbation.

<sup>1</sup> 'Medical Times and Gazette,' May 29th, 1875.

<sup>2</sup> Erichsen's 'Surgery,' 8th ed., edited by Beck, 1884, vol. i, p. 658.

<sup>3</sup> Holmes's 'Handbook of Surgery,' 2nd edit., 1878, p. 259.

<sup>4</sup> Billroth, 'Surgical Pathology' (New Syd. Soc.), 1877, vol. i, p. 324.

<sup>5</sup> Gross's 'System of Surgery,' 6th ed., 1881, vol. i, p. 1160.

Dr. Edmund Andrews,<sup>1</sup> in the article already quoted, thus expresses himself: "Resection of the head of the humerus is," he says, "a dangerous operation, but one which may be imperatively called for in cases where the axillary plexus is so compressed as to cause paralysis and every other resource has been invoked."

As regards the dangers of this operation I would with deference submit that they are not serious. The head of the bone may be deeply situated, and is doubtless surrounded closely by the vessels and nerves. The knife should therefore be kept in close contact with the bone, and should be laid aside in favour of a blunt raspatory whenever the structures seem to yield satisfactorily to the action of that instrument. It would probably also be unwise to have recourse to the operation, when much disintegration and hæmorrhage into the tissues, the result of recent attempts at reduction, are present.

But when all inflammatory processes have subsided, the operation may be undertaken with fair hope of sound and speedy union.

The operations designed and performed for the relief of old dislocations would therefore seem to fall under the following headings.

Firstly, cutting down upon the bone, and dividing those structures, muscular or ligamentous, which prevent the return of the head of the bone to its normal position.

Secondly, subcutaneous division of resistant structures.

Thirdly, subcutaneous division of the bone through the surgical neck.

Lastly, excision of the head of the bone.

Fracture may almost be placed out of consideration as a deliberate method to be advised and practised.

As regards the first of these methods, though doubtless it would be possible to restore the head of the bone to position by a sufficiently free use of the knife, the operation would be an extensive one, not devoid of risk, and should it succeed in design it is questionable whether the

<sup>1</sup> Ashhurst, 'Encyclopædia of Surgery,' vol. iii, pp. 671, 672.

displacement would not return, or a useful limb result. So far as subcutaneous division of resistant structures is concerned many of the same objections apply. In these cases, from various causes, the anatomical structures in the axilla may be altered in position, and indeed may be incorporated by inflammation with the very structures that need division.

Should firm bony ankylosis have ensued, the operation of subcutaneous section of the bone devised by Adams may prove advantageous.

The operation of excision of the head of the bone would thus be reserved for those cases of old dislocation where moderate efforts at reduction failed in accomplishing their object, and symptoms of pressure on the nerve-trunks and main vessels was present, or where great fixity and loss of movement existed, from neglect of passive motion by the patient and his attendants.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 354.)

ON THE  
NAKED-EYE AND MICROSCOPICAL VARIATIONS  
OF THE  
HUMAN THYROID BODY.

BY  
W. HALE WHITE, M.D.

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Received January 8th—Read March 18th 1888.

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THE object of the present paper is to try to discover how much the thyroid body may vary in persons who have not died from diseases in which, as far as our present knowledge goes, that organ is affected. The thyroid bodies taken indiscriminately from forty patients have been examined, and in order to prevent any bias I have not ascertained the disease from which the patient suffered till after the description of the thyroid was completed. Twenty-one were males, sixteen were females, and in three the sex was not stated.

In twenty cases the largest transverse section obtainable was taken ; the size of the section was found to vary very much, but owing to the different shapes, which were either more or less square, elongated, triangular or crescentic, the areas could not be measured.

The youngest patient examined was a male child aged

six months, the eldest a female aged seventy-seven years. Arranging the ages in a series according to the size of the transverse section, we find that the average age of the ten patients with the largest sections was thirty-eight years, of the ten with the smallest fifty-seven years. If we eliminate the child the youngest was twenty-eight years, then the average age for the larger glands is forty-two years against fifty-seven for the smaller. Thus it appears that the gland becomes smaller in old age. This is supported by the fact that one of the smallest was from the woman aged seventy-seven.

No connection could be discovered between the size of the gland and the disease from which the patient suffered.

Sometimes the vesicles are so minute as to be but little more than a ring of epithelial cells, and the whole section may contain nothing but these, whilst on the other hand there is hardly any limit to their size. The largest I met with were a tenth of an inch in diameter. It is not usual to find large and small vesicles both present in the same section, although to this there are many exceptions. A common size is  $\frac{1}{25}$ th of an inch in diameter. One does not find large vesicles in small glands, but one often finds small vesicles in large glands. I am quite unable to connect the size of the vesicle with any particular disease.

The type of the cells lining the vesicle is rectangular, taller than wide, with a large, oval, deeply-staining nucleus. Often they are quite flat, being considerably wider than they are high. This is so frequently associated with distension of the vesicle with secretion that the increased pressure seems to be the cause of the flattening. Often they are so flat that they are difficult to recognise. It is not easy to understand how secretion can have gone on under such an adverse pressure, but there are many instances in disease of a similar difficulty. When the vesicles are extremely distended they are often polygonal from the increased pressure. The contents of the vesicle have often shrunk away from its wall; some-

times they have taken with them the cells, usually called epithelial, which line the vesicle. I have not been able to determine the cause of this condition.

The epithelial cell takes the logwood stain poorly, but the nucleus, which has many nucleoli, takes it deeply. The nuclei are sometimes so large that they are contiguous and the cell-substance cannot be seen. The result is that the vesicle is lined by a number of darkly-stained oval bodies which look like leucocytes, but which from their regular arrangement, and from the presence of transition stages we must regard as nuclei; this is most often seen in thyroids having small numerous vesicles and but rarely when the vesicles are large and well distended.

I have, in a male aged thirty-four who died of heart disease, in a male aged forty-one who died of urethral stricture, and in a female aged fifty who died of cancer of the breast, seen a few vesicles having their epithelial lining involuted; sometimes the involutions are opposite so that a considerable constriction is caused. This has been noticed by several observers, and is usually looked upon as a persistence of the condition in which the gland was made up of a series of branching tubes.

Baber ('Phil. Trans.,' 1876, p. 563) has described parenchymatous cells which lie in groups between the vesicles. They are rounded and considerably larger than the epithelial cells; they are finely granular; each has a single nucleus staining darkly with logwood. Many of my sections show these cells. I cannot make out that the age, sex, or mode of death bear any relationship to the number of parenchymatous cells. My specimens support Baber's belief that these cells can make their way towards the vesicles through whose epithelial lining they may pass (Fig. 1) and thus become free in the vesicular cavity. When it reaches the colloid substance each cell forms for itself a concave depression. If a number of these are of equal size and arranged around the colloid matter it has the appearance of a toothed disc (Fig. 2). If they are more irregular it has a nibbled



appearance. This is very common and is best seen in medium-sized vesicles. It is never seen in the very large ones which are much distended with colloid material or in the very small ones which have hardly any. There are all gradations from this condition to that in which the vesicle contains nothing but granular *débris* with some nuclei scattered about in it, from which it would seem probable that the parenchymatous cells, having passed into the vesicle and destroyed the colloid matter, break up into granular *débris*. This view is supported by the fact that sometimes the parenchymatous cells will be seen to have lost part of their substance.

Sometimes there may be seen in the cavity of the vesicle bodies which are in all probability leucocytes that have wandered through the epithelial lining, but which it is difficult to distinguish from parenchymatous cells which have lost some of their cell-substance. While I think that Baber is right in his belief that leucocytes can pass through the epithelial lining I have never seen red blood-corpuscles in the vesicular cavity, but, from the brownish-red colour of its contents that one often sees, I should think that they also may find their way into the vesicle. The coarsely granular *débris* I have just described must be distinguished from the faintly granular appearance sometimes presented by the colloid matter itself. No one seeing the two conditions can possibly confound them.

As, in some vesicles in which there is much granular *débris*, the epithelium with which it is in contact is blurred or has even completely disappeared, it is possible that this granular *débris* may have been partly produced by the epithelium. The absence or altered appearance of the epithelium may, however, have been caused by the immigration of the parenchymatous cells.

I have roughly grouped my cases into those in which the granular condition was the striking characteristic, and those in which the large amount of colloid matter was the leading feature. There are seventeen in the first group and twenty-one in the second. I cannot make out any

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difference between the two classes as regards age, sex, or mode of death.

Baber suggests ('Phil. Trans.,' 1881) that the alcohol into which the thyroid is placed causes the colloid material to shrink and that the space thus left is filled by clear fluid which, exuding from the epithelium in drops, causes the indentation on the colloid matter. This hardly seems probable, for, as his figures and mine show, even when the indentations are most marked the teeth of colloid material may reach to the epithelium, so that we must suppose that the shrinking took place in an indented manner. Then again this fluid must be supposed to be quite different from the colloid material which is usually secreted by the epithelium, for it does not stain. For neither of these suppositions is there any reason. Further also, if the alcohol caused the shrinking it would affect all the colloid matter equally. It seems to me that at present we do not know the cause of the shrinking, but that the indentations are undoubtedly caused by the parenchymatous cells (Fig. 2).

Human colloid material appears to be exactly similar to that of the animals usually examined for histological purposes. It is opaque, either uniform and deeply stained by logwood or faintly granular and slightly stained. The shrunken appearance and toothed edge have been mentioned; often there are one or more large rounded holes in it (Fig. 3). In nine out of the forty thyroids examined these were numerous. Probably they are due to the invasion of the colloid material by parenchymatous cells, whose track cannot be seen either because it has closed up or the section is not in the right plane. This is supported by the fact that parenchymatous cells may be seen lying in the colloid matter (Fig. 4), and no doubt they frequently drop out in cutting the section. The size of the holes is easily explained by the power of the parenchymatous cells to absorb the colloid matter. Thus these holes and the indentations have the same origin.

The thyroids from two patients, one of whom died from

puerperal eclampsia and pneumonia at the age of twenty-one and the other from phthisis, show in some of the vesicles the colloid matter with a most remarkable double concentric arrangement, the centre part being a disc of homogeneous colloid matter outside which is a ring not quite so wide as the diameter of the disc of material to which it appears to be similar, save that it was darkly stained; both inside and outside the ring was a narrow clear space (Fig. 5). This condition has been described by Zeiss.

In a few specimens I have found crystals (Fig. 6) in the colloid material. They were best seen in a male, aged fifty-three, who died from a fracture of the base of his skull, and in a male aged twenty-seven, the cause of whose death was pericarditis; none of them were sufficiently distinct for one to be quite certain of the name to give them, but they might well be either cholesterin or oxalate of lime, both of which have been described as present in the colloid matter. Occasionally a hole, from which presumably a crystal has dropped out, may be seen.

The parenchymatous cells are often grouped together in small collections in such a manner that if each collection had a drop of colloid matter in its centre it would be very difficult to distinguish it from a minute vesicle. Sometimes one sees minute vesicles in which the lining cells are larger than usual, and are in most respects like parenchymatous cells. Roughly speaking it may be said that the larger the vesicle the smaller the epithelium, and that the smaller the vesicle the more the epithelium approaches the type of parenchymatous cells. For these reasons I would suggest that one method at least by which vesicles are formed is by the grouping together of parenchymatous cells, in the midst of which a minute drop of colloid matter makes its appearance. As this increases in size the epithelium decreases, till at last it is quite flattened out. Baber says ('Phil. Trans.,' 1876) the points of distinction between the parenchymatous and epithelial cells are that the former are large, more or less

rounded, the nucleus is darker, and does not stain quite so deeply with hæmatoxylin. These points are hardly sufficiently distinctive, and fig. 9 appended to his paper shows how parenchymatous cells may vary, and that some of them much resemble epithelial cells.

Some sections show large numbers of leucocytes scattered about among the interstices of the interstitial tissue. This condition, which was present in association with many very different diseases, was extremely well marked in a case of erysipelas.

Septa of fibrous tissue are prolonged into the gland from the fibrous sheath enveloping it; between these septa is a delicate connective tissue supporting the vesicles and vessels. In some thyroids the septa are very distinct, and split the organ up into islets, in others the supporting tissue is more uniform, and no arrangement of the vesicles into islets can be seen. When the vesicles are very large there is but little connective tissue between them; this is apparently due to its atrophy, owing to the pressure exercised by the distended vesicles. Some sections consist almost entirely of distended vesicles polyhedral from mutual pressure.

I have found, among the specimens I have examined, one in which the organ was nothing more than fibrous tissue (Fig. 7). It was quite as atrophic and degenerate as the thyroid in myxœdema. The patient, a man aged forty-five, had an aneurysm which pressed upon the recurrent laryngeal nerve. The report of the case makes no mention of any myxœdematous symptoms. This case is of the greatest value, for it appears to teach that in man the thyroid may be exactly like that found in myxœdema, and yet there may be no myxœdema present, but if no other instances of this are recorded I think we shall have to regard it as probable that this patient had myxœdematous symptoms which escaped recognition during life. In some other thyroids among my slides there was a considerable amount of fibrous tissue. One was from a man aged fifty-four, who died of granular kidneys and cancer of the

pancreas, and the other from a man aged fifty-four, who died from aneurism of the aorta which also pressed upon the recurrent laryngeal nerve; in this case the organ only weighed nine grammes. These three cases show that at present we very much want a more thorough examination of the thyroid in order to make quite certain that myxœdema is the only disease that is associated with atrophy of that organ.

In the 'British Medical Journal' (1885, vol. ii, pp. 342, 1014), founding my conclusion largely on the two cases here mentioned, in which the recurrent laryngeal nerve was pressed upon, I suggested that it might be the trophic nerve to the gland. As Mr. Victor Horsley has divided this nerve in dogs without producing any effect on the gland, we may conclude that probably this nerve has no trophic influence upon it.

The state of dilatation of the lymphatics varies enormously, and cannot be associated with any particular disease, but it is noteworthy that the only case of erysipelas examined showed greater dilatation than any other case.

The contents of the lymphatics consist, like that of the vesicles, of colloid matter, sometimes homogeneous, sometimes faintly granular. Holes may rarely be present in it (Fig. 8), and parenchymatous cells may lie in them. The coarse granular débris is but seldom to be seen in the lymphatics, and then in but small quantities. It may have a brownish-red tint.

Thus it appears that colloid material, parenchymatous cells, and granular débris can all be taken up from the vesicles by the lymphatics. The contents of the lymphatics never have a toothed edge. Occasionally a distended lymphatic is seen completely surrounding a vesicle.

I have observed nothing noteworthy about the blood-vessels.

I have not come across anything that is obviously an undeveloped portion of the gland, but it is, I should think, very difficult to distinguish small undeveloped portions from small groups of parenchymatous cells.

All my sections were prepared in exactly the same way, so that the difference observed cannot be set down to the method of preparation, which was to harden the gland in Muller's fluid and spirit, and then to stain the sections with logwood and mount them in Canada balsam.

My object has been to supply what seemed to be a want, especially as the thyroid is now so important pathologically, namely, a description of the size, shape, and microscopical appearances of the human thyroid when prepared by the ordinary methods. I think I have shown that in all these respects the organ may vary very considerably, and that we cannot at present see the meaning of these variations, which are probably none of them due to post-mortem changes and method of preparation. In passing, I have been able to point out that some unexplained appearances, which are very rare in the thyroids of the lower animals, may be present in man, and lastly I have tried to prove that the indentations of the colloid material are produced, not, as has been formerly supposed, by the exudation of drops of fluid from the epithelial cells, but by the migration of cells into the vesicles and also that new gland vesicles may originate in parenchymatous cells.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 357.)

## **DESCRIPTION OF PLATE I.**

**On the Naked-Eye and Microscopical Variations of the Human Thyroid Body. (W. HALE WHITE, M.D.)**

**FIG. 1.—**Parenchymatous cells forming depressions in the colloid matter, and wandering into it in large numbers.

**FIG. 2.—**The toothed-disc appearance of some of the colloid matter produced by the immigration of parenchymatous cells.

**FIG. 3.—**Holes in the colloid material.

**FIG. 4.—**Parenchymatous cells free in the colloid material of the vesicles.

**FIG. 5.—**The double contour arrangement of the colloid material.

**FIG. 6.—**A large crystal in the colloid material.

**FIG. 7.—**The fibroid condition of the thyroid in a patient who had not myxœdema.

**FIG. 8.—**Vacuoles in the colloid material of a lymphatic.

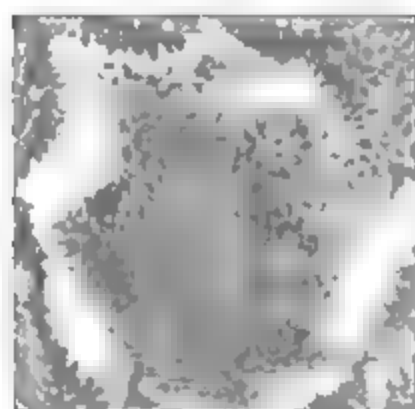


Fig. 1

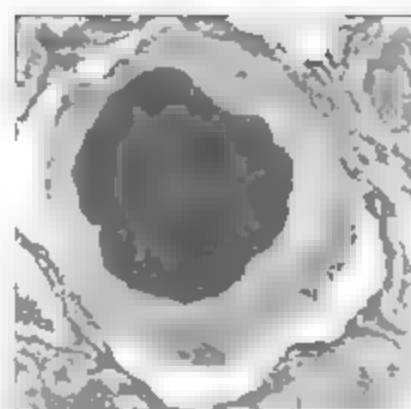


Fig. 5

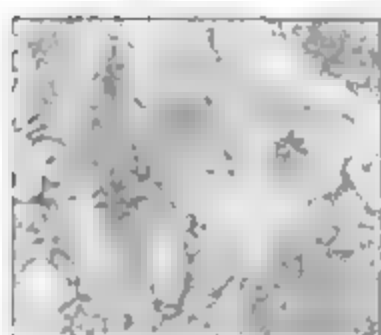


Fig. 2

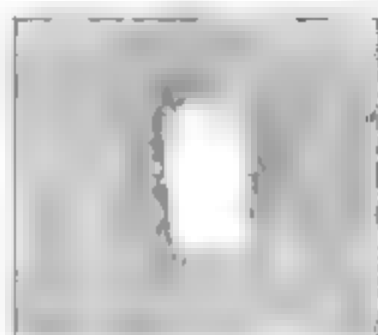


Fig. 6

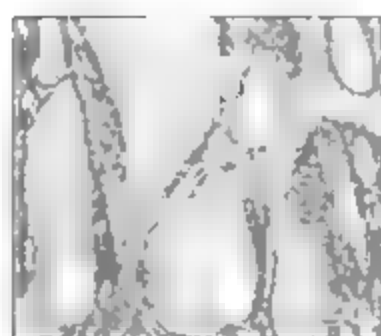


Fig. 3



Fig. 7

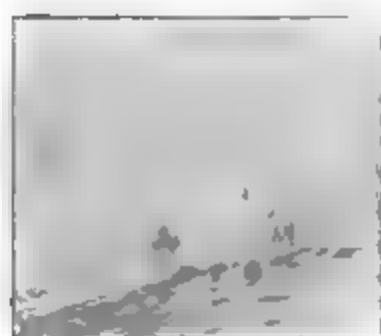


Fig. 4

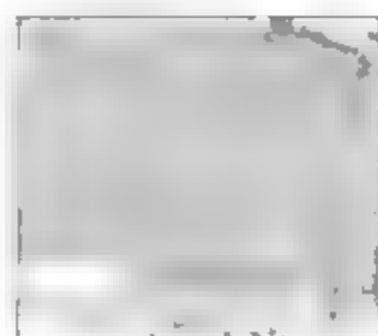


Fig. 8





A CASE  
OF  
EXTROVERSION OF THE BLADDER

TREATED BY PRELIMINARY NARROWING OF THE  
GAP EXISTING IN THE PUBIC SYMPHYSIS BY  
MEANS OF DIVISION OF THE SACRO-  
ILIAO SYNCHONDROSES.

BY  
G. H. MAKINS.

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Received December 10th, 1887—Read March 27th, 1888.

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IN the 'Centralblatt für Chirurgie' of December, 1885,<sup>1</sup> Professor Trendelenburg, of Bonn, published a case of extroversion of the bladder, in which immediate union of the lateral margins was obtained by a previous division of the sacro-iliac synchondroses. The operation attracted some notice at the time, and has since received the stamp of approval by Professor Thiersch,<sup>2</sup> one of the pioneers in the treatment of this defect, but as far as I know no surgeon in this country has repeated the method there detailed.

I feel I should make my apology to the originator of the method for bringing forward this case, since it is a

<sup>1</sup> 'Centralbl. f. Chir.,' No. 49, Dec., 1885.

<sup>2</sup> 'Verhandl. d. deutsche-Gesell. f. Chir.,' 1886, p. 13.

failure in one of the essential particulars, that of immediate union of the cutaneous margins of the incomplete bladder ; but although not a complete success it admirably illustrates the advantages which the preliminary operation offers, in contracting the area which has to be covered. I think, moreover, that all surgeons who have been called upon to treat cases of this nature must allow that much still remains to be wished for in the methods hitherto at our disposal. The case is as follows :

A. J—, æt. 5, male, one of seven healthy children. No hereditary history of malformations to be obtained, and the mother does not attempt to assign any cause.

The patient first came under the writer's care, by the kindness of Sir William Mac Cormac, in May, 1883. He was then one year and nine months of age, and his condition was as follows :

A fairly healthy-looking child with an extroverted bladder. The surface of the bladder is red and granular, in places encrusted with phosphates ; it measures about one and a half inches in diameter both from above downwards and from side to side. There are one or two excoriated patches encrusted with phosphates near the margin ; the surrounding skin is otherwise healthy. The penis is small and ill developed, complete epispadias exists, and there is a large pendulous prepuce. The scrotum is perfect, but both testes are undescended, and the inguinal rings are open. On the right side there is a tendency to hernial protrusion of the abdominal contents.

There is no umbilicus, but in the situation of the linea alba in its lower half is a shallow groove, bounding the margins of which are the recti abdominales, passing down to the unclosed symphysis, across which there is a gap of about three quarters of an inch.

On June 12th, 1883, chloroform having been administered, a flap of skin two and a half inches wide by five long was raised to thicken, with a view to covering the exposed bladder. Only a single flap was raised, although it was intended to raise a second one for the upper half

at a later date should the first flap do well. The operation unfortunately proved a failure, for on the sixth day the centre of the flap sloughed through. The resulting granulating surface quickly healed, leaving a cicatrix, which has proved the principal difficulty in all subsequent procedures. The child left the hospital in August, 1883, and was not seen again until his readmission on November 2nd, 1886, when he was brought to the hospital by his mother on account of some slight hæmorrhage occurring from the bladder surface.

The condition differed little from that existing in 1883, the only changes consisting in the greater size of the child, and the presence of a firm white cicatrix about one inch in length, which extended outward from the margin of the bladder on the left side, and was closely united to the subjacent aponeurosis of the external oblique. The scrotum was inflamed and red, and on its surface, as well as on the neighbouring skin of the thighs, were numerous ulcerated spots with raised margins, encrusted with phosphates, somewhat resembling mucous tubercles. The exposed bladder surface now measured three and a quarter inches in each direction. The patient was kept in bed twenty-seven days, and during this time the surrounding skin was got into a much better condition, and the bladder surface much improved by the application of pine-wood bags to absorb the urine as it passed.

On November 29th, 1886, the following operation was performed:—The bowels having been thoroughly cleared out during the previous day the patient was anæsthetised with chloroform. The space between the anterior superior spines of the ilia was measured, and found to be seven inches, that between the gaping symphysis one and a quarter inches, and the position of the great sciatic notch was determined by rectal examination. The boy was placed on his face, and incisions three inches in length were made over each synchondrosis. The posterior sacro-iliac ligaments were exposed and freely divided,

and the knife was then passed into the cleft, dividing the interosseous and superior ligaments, and also the interarticular cartilage. There was very little hæmorrhage. It was now found that slight pressure would allow the anterior superior spines to be approximated seven eighths of an inch, the joints gaping sufficiently posteriorly to allow the introduction of the forefinger.

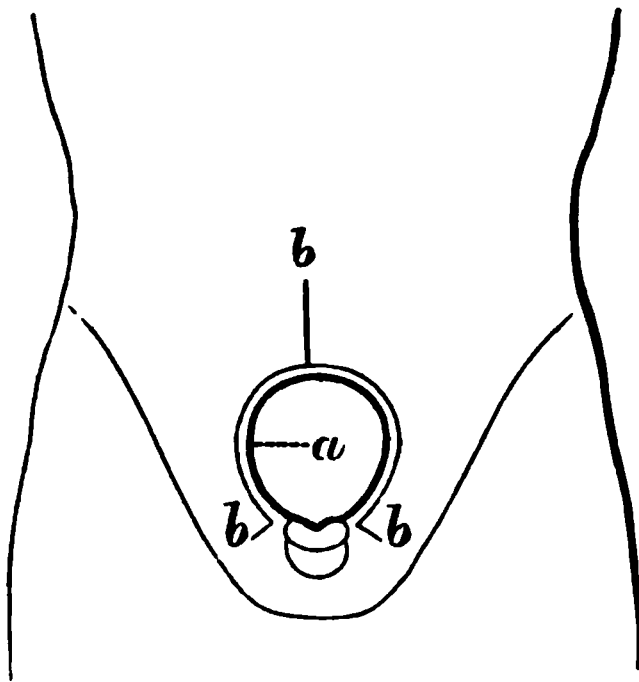


FIG. 1.—The outline, *a*, shows exposed posterior wall of bladder (when patient five years old), measuring  $3\frac{1}{4}$  in.  $\times$   $3\frac{1}{4}$  in; *b* shows line of incision made to connect margins.

Considerable pressure caused no further approximation, and this being deemed sufficient, the wounds were closed by suture, a split drain having been inserted at the lower angle. The surface was dusted with iodoform, some layers of iodoform gauze, covered with a stratum of salicylic wool, were applied, and the whole was covered in with a piece of thick gutta-percha tissue fixed to the skin around the edges of the dressing by chloroform. It was hoped that this would ensure the wound against the fouling which might result from the trickling down of urine. The surface of the bladder was covered with a pine-wood bag, which was to be removed as soon as it became soaked with urine.

The patient was then placed in his cot, and extension of the joints was provided for by placing him in a pelvic

belt, to the anterior borders of which three strips of strong webbing provided with loops had been sewn. These strips were crossed as a many-tailed bandage, carried over the opposite side of the cot, and there a piece of wood, to which a sandbag weighing 5 lbs. was appended was passed through the loops. Later in the day the bags were changed for others containing 7 lbs., and on the second day they were further increased to 10 lbs. on either side.

30th.—Comfortable. Temperature  $99^{\circ}$ . Some urine has found its way to the back. Dressing changed, as gutta-percha covering was giving way; some blood-stained serum in the gauze, quite sweet.

December 2nd.—Dressing changed, can kneel without much pain; skin around wound looking rather congested; left wound almost healed, so drain removed; right wound suppurating slightly. Sleeps badly but complains of no pain. Temperature  $98.6^{\circ}$ , has reached  $100^{\circ}$  twice. Tongue furred but moist. Appetite poor. Bowels not open since 30th. Signs of pressure over left anterior superior spine.

3rd.—Small slough over left anterior superior spine. The skin surrounding the bladder is lax, and the exposed surface is at least one third smaller than before the division of the synchondroses.

The patient continued to progress slowly; the wounds both gaped slightly and healed by granulation.

29th.—The sacro-iliac wounds, although still unhealed, are granulating healthily, the right being a linear surface only. The small slough over the left anterior superior spine has separated and the wound is granulating. The anterior superior spines are now six and one eighth inches apart, a gain of seven eighths of an inch. Does not suffer at all, and general condition is on the whole better than before operation, no doubt on account of the better nourishment he has been receiving. Kneels or can be shifted without any sign of pain.

January 22nd, 1887.—General condition good. Wounds

healed, except a small granulation on the left side. The exposed surface of bladder now measures only one and a half inches in each direction, and the anterior superior spines are one inch nearer than before operation. The decrease in the size of the exposed surface of the bladder

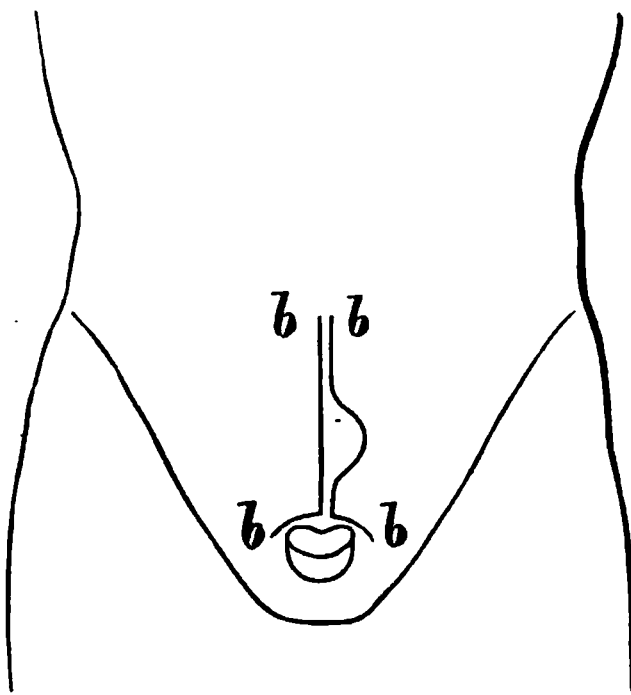


FIG. 2.—*b* Shows line of sutures.

is due not to mere contraction from relief of tension but to the formation of a deep groove on either side sufficient to contain some small quantity of urine as the patient lies on his back.

9.15 a.m., anæsthetised with chloroform. An incision, commencing one inch above the top limit of the bladder in the mid-line, was carried downwards on each side of the bladder to the lateral margins of the urethra; the skin was raised for about an inch and then drawn over the exposed bladder; the lower half was easily closed without the least tension, but the old cicatrix on the left side prevented the upper half meeting without tension, so that the skin had to be freed in an upward direction on that side for three quarters of an inch and then drawn down to fill the gap, the cicatrix being quite immovable. This portion of the operation is explained by diagrams 1 and 2. The lower margin of the abdominal skin was sutured to the margins of the penis, but no attempt was made to close the urethra.

The patient was again slung in his cot, and the flaps covered with a warm pine-wood bag, a large drainage-tube being inserted into the bladder from below.

The after-progress of this operation may be briefly disposed of; the skin became inflamed on the second day

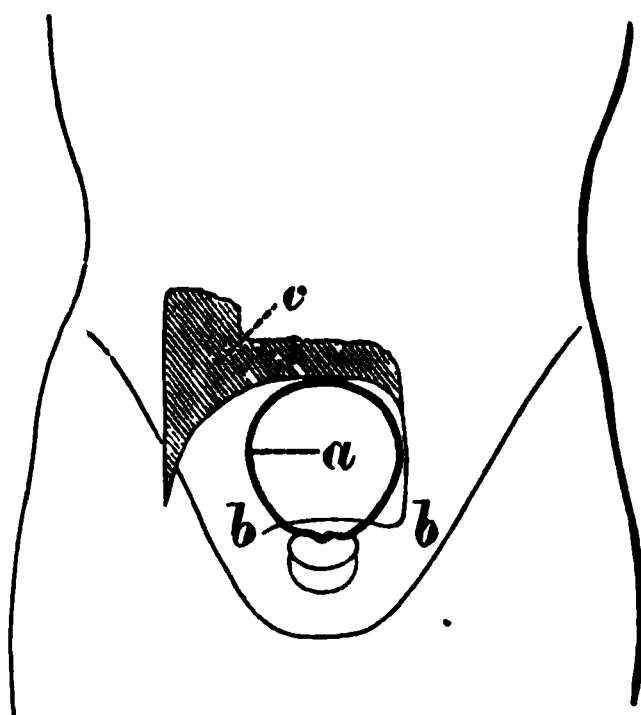


FIG. 3.—*a* Shows outline of exposed bladder at operation of May 4th, 1887; *b* shows large flap turned across and small flap turned down from upper margin; *c* shows raw surface.

and the whole gave way, some sloughing occurring at the edges. It is doubtful whether the warm pine-wood bags, which had been impregnated by soaking in a mercurial lotion, were not in some measure answerable for this failure. The granulating surface left rapidly healed, and forty-one days later it was decided to raise a skin flap on the right side. A flap, three inches by two, with its base at Poupart's ligament and its left edge close to the bladder margin, was lifted. This was allowed to thicken for two months, and on May 4th, 1887, it was turned across and united to the left margin, a small flap three quarters of an inch in width being turned down from above to close the upper limit of the cavity. The upper flap, however, consisted mainly of cicatricial tissue and sloughed, while the large flap, after holding for about ten days, became inflamed, and the wound suppurating, the bond of union completely gave way.



The wounds again cicatrised, and on June 16th he was sent to a convalescent home ; the flap projected from right margin of the bladder, and looked thick and healthy. The belt for compressing the pelvis had now been worn 198 days, and the space between the anterior superior spines was reduced by one inch and an eighth. He stayed at the convalescent home three months and three weeks, getting up during the day, and sleeping in a cot in the belt at night.

On September 29th he was readmitted, looking exceedingly well and bright. Walks firmly, and complains of no weakness in loins. The condition of the pelvis and bladder was as follows :—The line of the synchondroses is more palpable than normal as cicatrices dip down into them. The anterior superior spines are five inches and seven eighths apart, so that the gain of one inch and one eighth has been fully maintained. The exposed surface of bladder measures one inch in either direction, now amounting to less than one third of the superficial area previous to division of the synchondroses, and being less by half an inch than when it was first seen at one year and nine months of age. Surrounding skin thoroughly healthy in appearance, and the flap made for the last operation shifts readily on the subjacent structures.

October 27th, 1887.—Patient anæsthetised with chloroform. The old Thiersch's flap raised, and a small flap three quarters of an inch in width raised on the left side of the bladder.

The small flap was turned with its cutaneous surface towards the bladder, and three sutures were passed from the margin of the incision beneath the raw surface, emerging at the base of the flap just at the margin of the bladder. The large flap was now turned over and the three deep stitches made to pierce it just to the left of the centre. The refreshed end of the large flap was fastened by a deep and superficial row of fine sutures to the raw surface made by raising the small one, and finally the deep sutures were twisted so as to hold the whole in

apposition. A small incision was made across the old cicatrix to relieve tension, but no attempt was made to connect the lower margin of the flap with the penis. A

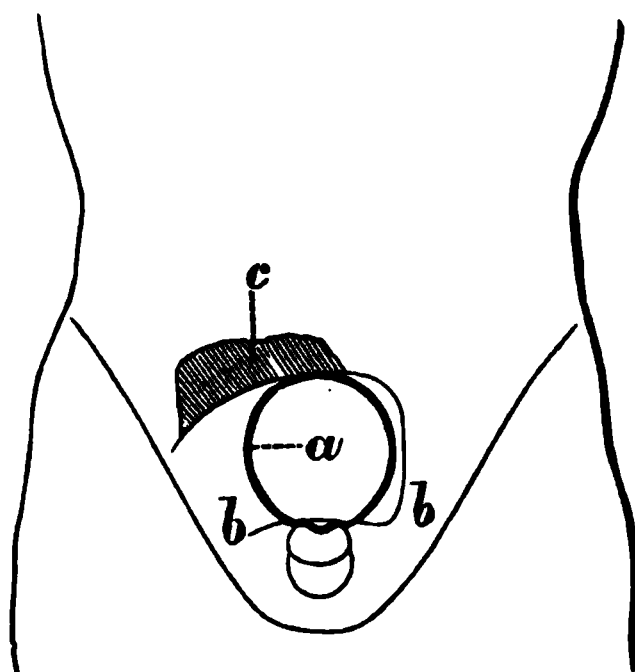


FIG. 4.—Shows flap turned across on Oct. 27th, 1887. The shaded part, *c*, shows raw surface.

piece of oiled lint was placed over the raw surface, but the flaps were left uncovered, a cradle containing a hot bottle to heat the air being placed over the patient.

The flap healed perfectly, entirely covering the exposed bladder, but leaving a gap one inch and a quarter long at the upper limit, through which the mucous membrane could be seen on holding the surfaces apart. This gap was bounded at each end by cicatricial tissue, but above and below by skin, and on November 28th the edges were refreshed, and the opposing surfaces sutured.

The gap at the upper border of the bladder has since been closed, except a small sinus, but any further operation in the direction of forming a urethra is for the present deferred on account of the troublesome disposition to phosphatic deposit.

I will shortly point out the advantages which seem to belong to this operation, especially in their bearing on my case, and answer as far as I am able the only objection which seems likely to be raised.

The operation is necessarily somewhat limited in appli-

cation, since the consolidation of the synchondroses would render it impracticable in any but young subjects. Prof. Trendelenburg originally placed the limit between two and four years, but has since raised it to five, and in a private letter I have received from him he says he now considers five the most suitable age.

1. In favorable cases a very great saving of time is effected. In one of Trendelenburg's cases the whole procedure, excepting the closure of a small fistula, occupied only some eight weeks.

2. In cases where complete success is attained, the margins of the bladder being approximated with the skin an ideal result is attained, the lining membrane, with the exception of the anterior linear cicatrix, being formed by the actual vesical mucous membrane, and irregular cicatricial pouches are avoided. As a consequence, the probabilities of phosphatic incrustations, or the formation of calculi, is greatly lessened.

3. Failure of the primary operation in no way prejudices further measures such as the raising of flaps.

4. Should flaps be subsequently needed, as in my case, the area to be covered is so much decreased in size that the procedure is easier and more likely to be followed by success, since less than half the ordinary amount of skin is needed.

5. The superficial area is not only lessened, but an important preliminary to the formation of a receptacle is developed in the sinking backward of the bladder wall, especially as a deep groove around the margin.

6. This lessening of the area to be covered especially adapts the method to cases such as mine in which cicatrices already exist and render the lifting of bilateral flaps difficult or impossible.

7. The closure or approximation of the symphyseal gap affords a better support for the abdominal viscera, while the rotation of the ilia corrects in some measure the acquired rotation outwards of the lower extremities often noted in these patients.

8. Lastly, Prof. Trendelenburg expresses the hope that in some of these cases the sphincter apparatus may still exist, and that by union of the posterior portion of the urethra a fairly retentive viscus may be obtained.

The chief objection no doubt lies in the possible weakening of the joints. On this matter I can only say that my own patient walks well, and that in Prof. Trendelenburg's cases the same result has been noted.

I would point out, however, that although the posterior, interosseous, and superior ligaments are divided, that the ilio-lumbar and sacro-sciatic ligaments remain entire. The latter would still be equally able to resist possible rotation of the sacrum forwards, the ilia being connected and fixed above by the ilio-lumbar ligaments to the transverse process of the last lumbar vertebra, and moreover the slightly increased forward obliquity of the latter ligaments would tend to prevent displacement of the sacrum backwards, or movement of the ilia forwards; the latter rotating forwards in the movement approximating the anterior superior spines on the combined axes of this ligament and the anterior margins of the joint.

It must be remembered also that during the maintenance of the compression, although the posterior aspects of the joints are caused to gape, yet the compression is lateral, and consequently the irregular surfaces offered by the opposing bones would still tend to prevent gliding movements in either direction, the division of the joint-union having of necessity followed to a considerable extent these inequalities.

As to the mode of repair, the gap must at first necessarily become filled with soft connective tissue, and for the fulfilment of this object slight suppuration, such as occurred in my case, is perhaps to be regarded as favorable. I would point out, however, that the result of the compression is to relieve the ossifying structures bounding the posterior aspect of the joint from pressure, while pressure is considerably increased in the anterior segment. Reasoning from the analogy of what is observed in the

case of other growing bones when pressure is exerted on one portion of the ossifying cartilage, as in the treatment of genu valgum by splints, or the deformity of the upper surface of the astragalus developing in talipes equinus, I think it may be regarded as probable that the bone bounding the posterior part of the gap may develop more extensively than that of the anterior portion, and hence that the fissure may eventually be little wider than a normal one.

It has also been objected that the method is inapplicable to females (Sonnenburg) since it would no doubt materially contract the pelvic openings. Prof. Trendelenburg writes me that he has lately successfully operated on a female child, and the case will be published at the next German Surgical Congress. In any case, however, the objection is the less important from the comparative infrequency of the condition in females.

In my own case one or two particulars still require mention. In the operation I contented myself with division of the greater portion of the joint, not breaking the two sides free, as has been done by Prof. Trendelenburg. My reason for this was that the amount of division allowed approximation of the spines one inch with moderate pressure, and as a result of trial I found very considerable force would be needed to entirely separate the joints, while I had already passed the knife as far forwards as I felt was safe. With entire freeing of the joints Prof. Trendelenburg has, however, gained as much as two inches in a child of two and a half years. In connection with this point mention should be made of the proposal of Dr. Hirschberg to apply compression of a similar character to the pelves of young infants without any division of ligaments, a method which in the light of my own case certainly deserves a trial.

The very great decrease of the area to be covered has already been mentioned, the exposed surface being considerably smaller than it was three and a half years previously. I would mention here also a minor point with regard to

the treatment of the bladder surface. When first admitted it was granular, easily bled, encrusted with phosphates, and small ulcers existed on the skin of the thighs, scrotum, and abdomen. Washing with antiseptic lotions and dilute nitric acid caused very little improvement, but after the application of the pine-wood bags, impregnated with perchloride of mercury, the surfaces rapidly cleaned, healed, and remained sound.

Lastly, the operation has been performed by Trendelenburg in at least five cases, four males and one female. In one of these cases a failure occurred at fourteen months of age, but the operation was repeated at three years of age successfully. In only one of the five cases had flaps to be resorted to, and in two the result was typical success.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 359.)



**A CASE  
OF  
WOUND OF THE FEMORAL ARTERY  
AND VEIN.**

**TRAUMATIC VARICOSE ANEURISM; LIGATURE OF BOTH  
ARTERY AND VEIN; RECOVERY.**

**WITH REMARKS ON THE TREATMENT OF WOUNDS OF  
THE FEMORAL ARTERY AND VEIN.**

**BY**

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R. H. W—, æt. 19, a medical student and surgical dresser at St. Bartholomew's Hospital, was warded on January 27th, 1885, for a punctured wound in the upper third of the left thigh. He was preparing for a small operation in the ward, and was pulling out a drawer from the box containing the instruments, when the drawer, which worked stiffly, came out with a jerk. Several knives in their fall stuck in his trousers, and one was driven some distance into the thigh by the drawer striking against its handle. He was immediately placed on a bed and the hæmorrhage controlled by digital pressure, and subsequently by a pad and bandage, but before this was accomplished he lost about eight ounces of blood. On removing his clothes two wounds were discovered in the upper third of the thigh near the apex



of Scarpa's triangle, one wound being a little above the other, and the lower one appearing quite superficial. Each measured about half an inch in length, with its long diameter transverse.

As digital pressure on the upper wound, and pressure three quarters of an inch below it, completely controlled the hæmorrhage, whilst pressure on the femoral above failed to do so, a wound of the long saphenous vein was diagnosed by the house surgeon, Mr. Lankester, and a graduated compress was placed over the wound, and the limb firmly bandaged from below upwards. On the following day, January 28th, at the afternoon visit of Mr. Walsham, who was on duty for Mr. Willett, the bandages were removed and pulsation was seen to be communicated to the pad covering the wounds, the edges of which were found to be adherent when the pad was taken off. A soft distensile pulsating tumour, about the size of a small orange, was now discovered to the inner side of the two wounds. The pulsation was controlled, though not absolutely, by compressing the femoral artery above. A distinct bruit was heard along the course of the artery, and a venous hum could be heard and a thrill felt along the course of the femoral vein. A wound of the femoral artery and vein with the formation of a traumatic arterio-venous aneurism was diagnosed, and a consultation was held. It was decided that no operative interference should be undertaken at present on account of the excited state of the patient's circulation, but that firm pressure should be applied both over the tumour and along the course of the artery. A pad was accordingly placed over the tumour, and two pieces of firewood about six inches long, well protected with lint, over the course of the artery both above and below the tumour. The whole limb was then firmly and carefully bandaged from the foot upwards, the bandage terminating in a spica at the groin. The limb was raised and supported by pillows. Fifteen grains of bromide of potassium were given every six hours.

January 29th.—The patient passed a good night; the tumour was thought to be somewhat smaller, and the venous hum less marked. His temperature remained normal, but his circulation was still in a very excited state; the pulse could be felt almost everywhere on touching the limb.

On the 30th, the tumour continued in much the same condition, but on removing the bandages on the 31st it appeared to be nearer the surface, the pulsation was more marked, and the wound in the skin, which had become sealed, seemed likely to give way. Mr. Walsham decided, therefore, to operate at once, in which decision he was supported by his colleagues, who saw the case with him in consultation.

*Operation.*—The patient being placed under an anæsthetic, and the femoral controlled by digital pressure, a probe was passed into the upper wound, and entered a cavity containing effused blood. On enlarging the wound it was found that the digital compression did not sufficiently control the hæmorrhage, and the india-rubber tube of the Esmarch's apparatus was accordingly applied to the upper third of the thigh, and after a considerable quantity of effused blood had been turned out of the cavity a wound was discovered in the femoral artery in the middle of Hunter's canal. The wound was about three quarters of an inch long, and ran obliquely to the long axis of the artery. A ligature of kangaroo-tail tendon was placed above and below the wound; and the vessel divided between the two ligatures. Slight hæmorrhage continued after the artery had been tied, but no wound in the vein could at first be found. The Esmarch's bandage was accordingly temporarily loosened, but had at once to be re-tightened on account of the immediate increase of hæmorrhage. After considerable difficulty a wound in the vein was discovered; it ran obliquely along the course of the vein, and was nearly an inch in length. The vein was secured above and below the injured spot by two kangaroo-tail tendon ligatures, but not before a

piece of the artery between the ligatures had been cut away to gain sufficient access to the vein. On the removal of the Esmarch bandage the hæmorrhage was found to be effectually stopped, and the wound having been thoroughly cleansed of all clot and well irrigated with carbolic lotion, was brought together by silver-wire sutures and dressed with carbolised gauze, and a drainage-tube inserted. The operation, which was done under the spray, took nearly two hours. The limb was wrapped in cotton-wool, surrounded with hot bottles, and raised to an angle of  $120^{\circ}$  with the body. The patient was ordered tincture of opium  $\text{m}\nu$  every two hours whilst awake.

On the following morning the note states:—Passed a fair night, temp.  $102^{\circ}$ ; the foot is warm, and retains perfect sensibility. The pulse in the posterior tibial artery is perceptible. There is no œdema. The discharge had come through the dressings at one spot; the dressings were not removed, but fresh layers of gauze applied.

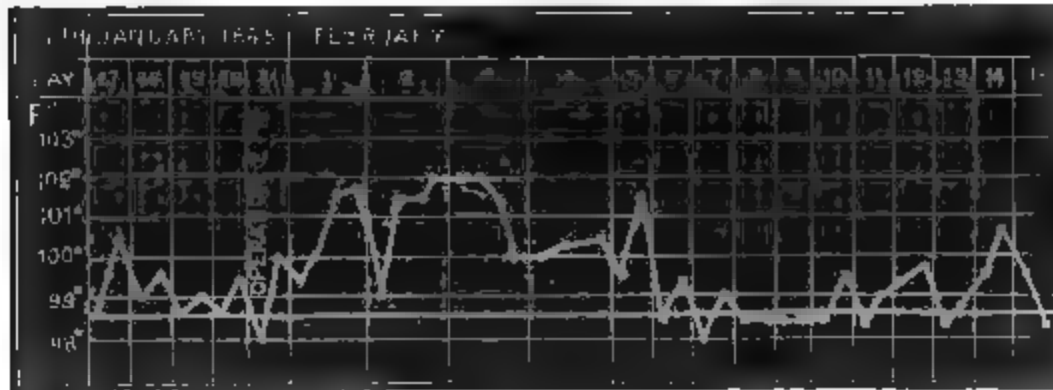
On February 2nd, the wound was dressed; looking well; edges united by first intention, except in situation of drainage-tube. The condition of foot perfect.

On the 3rd, the wound was dressed and all stitches were removed; the edges at the lower part had given way. From this time the patient made an uninterrupted recovery, but a sinus in the situation of the drainage-tube remained for several weeks, and did not finally close till the latter end of March. The temperature on the evening of the day after the operation rose to  $101.8^{\circ}$  and on the next evening to  $102^{\circ}$ , but from this date fell to a little above normal, and continued practically normal from the sixth day. (See Temperature Chart.)

The greater part of the after-treatment was conducted by Mr. Willett, to whom my thanks are due for allowing me to publish the case.

Wounds of the femoral artery and vein are sufficiently rare, I venture to think, to make this case of considerable interest; and when taken in conjunction with the fact

that both the artery and vein were ligatured renders it, I hope, worthy of bringing before the notice of the Fellows of the Royal Medical and Chirurgical Society.



As the upper wound was situated in the course of the long saphenous vein, and pressure below and on the wound stopped the hæmorrhage, whilst pressure on the femoral artery had no effect, it was supposed that the long saphenous vein had been wounded, and it was not until the removal of the bandage on the afternoon of the following day that the grave nature of the injury became evident. The presence of a deep pulsating tumour in the situation of the wound, and the well-marked arterio-venous thrill and bruit then detected, made it certain that the femoral artery and vein had been wounded, and that a communication existed between them. The question at once arose, Should immediate steps be taken to secure the bleeding artery and vein, or should carefully applied pressure be first tried? In favour of the former plan it was urged that here was undoubtedly a wound of a large artery, and that the sooner it was secured the less danger there would be of further extravasation of blood and tearing up of the tissues, and that all risk of a sudden hæmorrhage from the giving way of the wound would be effectually avoided. On the other hand, it was evident that the femoral vein as well as the artery was injured, and might probably require ligature—a step that would add to the danger of the operation. Moreover, the superficial wound had become sealed; there was apparently a

considerable thickness of tissues between the surface and the extravasated blood, and some form of a sac, as evidenced by the pulsation, had already been formed by the condensation of the fasciæ and other tissues around. It was therefore decided to try the effects of pressure, and it was thought, moreover, that should this fail there would be less danger (in consequence of the collateral circulation having had time to become at least to some extent established) of gangrene supervening if both the artery and vein should subsequently require ligature. The further question was also raised—What, in event of the artery having to be tied, should be done with the vein? Should it be tied above and below the wound in its walls? Should the artery only be tied, and pressure applied to the vein, or should, supposing the hole in the vein to be small, the wall be nipped up and a ligature applied without obliterating its calibre?

I need not repeat what has already been said in the history of the case. Pressure failed, the artery was tied, and the rent in the vein was of such a nature that nothing was left but to apply a ligature to it above and below the wound. Before the operation was undertaken, however, I was considerably exercised in my mind as to which of the above methods of procedure it would be the best to adopt, and some difference of opinion was expressed by my colleagues who kindly saw the case with me in consultation. Since then I have made a somewhat extensive search through the literature of the treatment of wounds of the femoral artery and vein, and I venture to bring the question of the best treatment of such before the Fellows of the Society. I propose to discuss the question under the following heads:

1. Immediate simultaneous ligature of the artery and vein.
2. Continuous pressure without operation.
3. Temporary pressure in order to allow the collateral circulation to become established before resorting to ligature.

4. Ligature of the artery and application of pressure to the vein.

5. The question of the lateral *versus* the circular ligature of veins.

### 1. *Immediate simultaneous Ligature of Artery and Vein.*

When the wound of the soft parts leading to the injured vessels is large, death, I imagine, as in four of the cases tabulated in the appendix to this paper, will probably occur in a few minutes. To such the question of the propriety of immediate ligature obviously does not apply. Were such a case seen before death had actually occurred, ligature of both the artery and vein or amputation would be clearly the only course. In the case of a punctured wound, however, where the hæmorrhage can be controlled by pressure, it is not so evident that immediate ligature is the right treatment to pursue. Langenbeck,<sup>1</sup> as is well known, recommends that in wounds of the main vein the accompanying artery should be tied as well as the injured vein. He affirmed that when both vessels are tied not only does gangrene not follow, but there is less disturbance to the capillary circulation than when the vein or artery alone is secured. By the simultaneous ligature, he believed, an equilibrium is maintained between the arteries and veins until the collateral circulation is established.<sup>2</sup>

<sup>1</sup> 'Archiv klin. Chir.,' 1860, Band i, p. 543.

<sup>2</sup> Further, it has been shown (Cohnheim, 'Vorlesungen ueber Allgemeine Pathologie,' Bd. i, p. 122) that if in rabbits the main vein in one limb is tied, and in the other both the vein and artery, in the latter case there is no disturbance of the circulation, whilst in the former swelling and œdema occur. The condition of the capillary circulation under these circumstances has been demonstrated microscopically in the web of the frog. In the limb in which the vein only is tied the veins and capillaries are greatly dilated and congested, and the blood either circulates very slowly or complete stasis occurs, whilst oscillation of the blood current is observed in the arteries. On the other hand, in the limb in which both artery and vein are ligatured, although some of the capillaries may be found empty and the blood in them

The view of Langenbeck, however, that there is less risk of gangrene when the main vein is tied as well as the main artery, in that a smaller quantity of blood is conveyed to the part beyond, and less stress is thrown upon the over-charged vein, is not generally, I believe, accepted by surgeons, and was controverted at some length in a leading article in the 'Lancet'<sup>1</sup> of last year. The ligature of both vessels, it is there stated, has been successful in certain cases; but, on the whole, the mortality and the frequency with which gangrene has occurred have been greatly increased by this practice.

Ligature of the main artery, it is urged, is attended with two results which are unfavorable to the venous circulation; in the first place the local anæmia causes a general dilatation of the arterioles with compression of their attendant venules, and in the second place the force of the heart is dissipated in propelling the blood through the many anastomosing arteries. Moreover, Pirogoff,<sup>2</sup> Stromeyer,<sup>3</sup> and Spence,<sup>4</sup> among others, taught that sudden obstruction of both the artery and vein almost invariably leads to gangrene; and Braune,<sup>5</sup> from experiments on the dead body, concluded that when the common femoral artery and vein were suddenly occluded gangrene must inevitably ensue. On the other hand, Mr. Annandale,<sup>6</sup> in speaking of a case in which he tied the main artery and vein for arterio-venous aneurism, says, "The result of the operation in my case tends to prove that surgeons have taken too serious a view of the

at rest, there is no venous stasis and engorgement, and oscillation is not observed in the arterioles, whilst in other parts the circulation is carried on in a normal manner.

<sup>1</sup> 'Lancet,' Feb. 13th, 1886.

<sup>2</sup> 'Grundzüge der allgemeinen Kriegschirurgie,' vol. i, p. 440 (Leipzig, 1864).

<sup>3</sup> Stromeyer, 'Handbuch.'

<sup>4</sup> 'British Medical Journal,' 1875, vol. ii, p. 195.

<sup>5</sup> 'Die Oberschenkel Vene des Menschen in anatomische und klinische Beziehung (Leipzig, 1871).

<sup>6</sup> 'Lancet,' April 24th, 1875, p. 568.

risks likely to follow the ligature or obstruction of the principal artery and vein of the limb." The cases that have now been published and are collected in the accompanying tables go far, I think, to show that in this opinion he was probably correct. A distinction, however, must be drawn between immediate ligature of the main artery and vein for a wound of one or both of these vessels, and their deligation for aneurism or for a wound in the removal of a tumour where in either case, as the result of previous compression, the collateral circulation has had time to become established. And, further, account must be taken whether the vessels tied are the common femoral or the superficial femoral or popliteal. Thus, in twelve cases (see Table II) in which both artery and vein were tied immediately after the wound, gangrene is said to have occurred in four cases, and probably occurred in a fifth, as amputation in this latter was performed subsequent to the ligature. The remaining cases, except one<sup>1</sup> in which the cause of death is not stated, recovered without gangrene.

The above are the only instances I have been able to find in which the femoral artery and vein have been simultaneously ligatured for a wound of these vessels, without pressure for some days having been previously applied, and granting that they are too few to allow any reliable conclusions to be drawn from them, still they show, I think, that although there is a considerable risk of gangrene, especially when the common femoral is tied, this risk has been over-estimated.

<sup>1</sup> In this case it appears probable that gangrene of the leg would have ensued if the patient had not died of other causes, as there was loss of sensation in the leg, and it did not bleed on puncture. The patient, who was a heavy drinker, became delirious on the fourth day, and vomited violently.



## 2. *Continuous Pressure without Operation.*

Where the communication between the artery and vein is at all large, the chances of success from this method appear to be very small. In the case under consideration the tumour steadily increased, notwithstanding that careful pressure was applied; and an external hæmorrhage, if the wounded vessels had not been secured, would inevitably have occurred in a few days. In the thirty-six cases that I have collected and tabulated (Tables III and V) in none was this method completely successful, inasmuch as in all an arterio-venous aneurism formed, and only one of these aneurisms<sup>1</sup> was cured by the continuance of the pressure. In another case<sup>2</sup> pressure so far succeeded in that the aneurism became partially consolidated, but an untimely examination displaced the clots and ligature had to be resorted to. Of course it may be argued that in these cases pressure was not efficiently employed, and in some no doubt it was not. But in many, as in my own case, it is specified that every attention and care was observed, and although in others full particulars are not given, there is reason to believe that no pains were spared. In all of the cases, with the exception of the two mentioned above, operative treatment of some kind had ultimately to be undertaken, or such palliatives as elastic compression to be employed. It is true that in some instances the

<sup>1</sup> In this case Trélat succeeded, by long-continued instrumental pressure, in converting an arterio-venous aneurism, the result of a punctured wound of the femoral artery and vein, into a simple arterial aneurism; and this was subsequently cured by a continued application of pressure. Nélaton has succeeded four times in converting a varicose aneurism at the bend of the elbow into a simple arterial aneurism by direct pressure (Holmes's 'System of Surgery,' vol. iii, p. 92).

<sup>2</sup> In Mr. Coote's case the aneurism had become consolidated and pulsation had ceased, but owing to the tumour having been examined somewhat prematurely, and perhaps roughly, by visitors, the clots were disturbed, pulsation recommenced, and the artery and vein had to be tied. Death occurred after amputation from secondary hæmorrhage ('St. Barth. Hosp. Rep.,' 1874, vol. iii, p. 544).

inconvenience from the arterio-venous aneurism at the time the case is reported was very slight, as, for instance, in the cases published by Messrs. Thomas Smith,<sup>1</sup> Hulke,<sup>2</sup> and Pick<sup>3</sup>, but in others, the patient's state was such a miserable one that he was willing to consent to any operation, whatever the risk.

Further, even in cases where soon after the injury the inconvenience is slight, it is questionable what the ultimate condition will be. Thus in Mr. Cock's case<sup>4</sup> it was two years before the superficial veins were dilated, but still surgical interference became necessary, although eleven years had elapsed. Again, in Beaumont's case<sup>5</sup> the communication between the artery and vein existed for ten years and a half before a tumour formed by the giving way of the weakened artery, but an operation then became imperative. "The treatment of arterio-venous aneurism," says Mr. Erichsen,<sup>6</sup> "is exceedingly unsatisfactory," a statement which I believe is endorsed by most surgeons who have had these cases under their care, and is fully borne out by the cases published in Tables III and V. An operation of some sort was performed in thirty-one out of the thirty-six cases there collected and seventeen died. So impressed with the difficulty and danger of the treatment of arterio-venous aneurism, especially of the femoral artery and vein, is M. Polaillon<sup>7</sup> "that the formation of one," he says, "ought in all instances to be prevented by immediate ligature wherever it is suspected that a communication between these vessels exist." "If the aneurism is already formed," he continues, "we should act as soon as possible, for in temporising great and perhaps insurmountable difficulties may be met with. If, therefore, it is a fact that the only

<sup>1</sup> 'St. Barth. Hosp. Rep.,' vol. xii, p. 157.

<sup>2</sup> 'Clinical Society's Transactions,' vol. viii, p. 175.

<sup>3</sup> 'Med. Times and Gazette,' 1883, vol. ii.

<sup>4</sup> 'Med.-Chir. Trans.,' 1851.

<sup>5</sup> 'Med. Times and Gaz.,' July 17th, 1867.

<sup>6</sup> 'Science and Art of Surgery,' 8th edit., p. 463, vol. i.

<sup>7</sup> 'Gaz. Méd. de Paris,' 1886, vol. iii, p. 265.

result of pressure without subsequent operation is to leave in the great majority of cases the patient with an arterio-venous aneurism, the difficulties and dangers of treating which increase in consequence of the dilatation of the artery and vein the longer operative treatment is delayed, it follows, I think, that pressure should not be continued for more than a few days unless signs of evident improvement ensue—in short, that pressure should only be kept up till the collateral channels have had time to enlarge.” But of this more under the next section. Further, pressure long continued is in itself not unattended with danger. Thus in Case 7 (Table V) the pressure caused cellulitis for which incisions had to be made, and the patient succumbed to septicæmia; whilst in Ward’s case (Table III), after nine days of pressure secondary hæmorrhage occurred, and the artery and vein had to be tied in the midst of a sloughy wound.

3. *Temporary Pressure till the Collateral Circulation has had time to become established before resorting to Ligature.*

Although, as has been shown, there is considerable risk of gangrene of the limb occurring when the main artery and vein are suddenly occluded, this risk appears to be reduced to a minimum when, owing to partial obstruction of a vein, either from pressure applied by the surgeon, or pressure produced by the growth of a tumour, the collateral vessels have become enlarged. Thus Grillo,<sup>1</sup> of Naples, tied the femoral vein with the femoral artery in fifteen cases of aneurism without a single bad result, and Hunter, as is well known, in his first three operations tied the artery and vein by a common ligature. In twenty cases here tabulated (Table III) in which the artery and vein were tied for a wound of one or both of the vessels, but not until some days or even some months

<sup>1</sup> Lidell’s article in Ashhurst’s ‘International Encyclopædia of Surgery,’ vol. iii.

or years had elapsed, gangrene occurred in only five cases; and in four other cases (Table IV) in which the vessels were tied in consequence of a wound in removing a tumour of the thigh pressing on the vessels, no gangrene ensued; and in two of these, and probably in three, the ligatures were applied to the common femoral trunks. In four out of the five cases in which gangrene did occur a satisfactory explanation is forthcoming; in the fifth the details are too meagre to argue either one way or the other. Thus in Case 12, Table III, the collateral circulation had not become established notwithstanding the time (nine days) that had elapsed since the injury, for although the limb was warm at the time of the operation the pulse was not felt in the tibials, and subsequently, at the post-mortem examination, the profunda artery was not found to have enlarged, and the profunda vein was plugged.<sup>1</sup> In the second case (No. 13, Table III) the gangrene was due to the compression of the common femoral vein by the accidental slipping of a clamp which had been placed on the femoral artery above the ligatured end as a precaution against secondary hæmorrhage. In the third case (No. 17, Table III) it was necessary to tie the profunda artery as well as the femoral, and at the post-mortem examination the long saphenous vein was found plugged. In the fourth case (No. 20, Table III) secondary hæmorrhage occurred subsequent to the ligature of the superficial femoral vessels, for which the common femoral was tied. Besides the above twenty cases Mr. Holmes, in his "Lectures on Aneu-

<sup>1</sup> This fact is explained on the supposition that most of the blood brought to the divided femoral extremity found its way back to the divided femoral vein; that the pressure was thus relieved, and that the profunda received only its usual supply of blood. For three days there was no sign of interference with the circulation, but at that time gangrene appeared and spread with great rapidity. This was explained by the recent phlebitis of the profunda vein, which was filled with a pink firm coagulum. The inflammation of this vein, so vital to the support of the circulation, was probably the result either of the irritation of the operation or injury by the ball ('Med. and Surg. Hist. War of the Rebellion,' vol. ii, part iii, p. 213).

rism,"<sup>1</sup> refers to a case in St. George's Hospital Tables, in which the femoral vein was tied as well as the artery, and yet no gangrene ensued; and further, to a case of Mr. Perry's of arterio-venous aneurism, in which the femoral vein was plugged with coagula, and the profunda vein was so compressed as hardly to transmit any blood; still the trunk veins anastomosing with the saphenous had so completely carried on the circulation that there was not only no gangrene but no cedema of the limb. From a consideration of these cases I think it is evident that gangrene, which appears to be the chief risk after immediate ligature, is reduced to a minimum when, as the result of carefully applied pressure, the collateral circulation has become established. But it has been shown in the former section that pressure in itself holds out little chance of ultimate success, and at the best will merely leave the patient with the external wound healed but with a permanent arterio-venous aneurism. If these premises are sound it follows that the safest treatment in punctured wounds of the thigh implicating the femoral artery and vein is to control by pressure the circulation through the wounded vessels for a few days until the collateral channels have become enlarged, and then to cut down upon and tie the artery and vein above and below the wound.

#### *4. Ligature of the Artery and application of Pressure to the Vein.*

In the case now before the Society the wound in the vein was so extensive that it was at once evident that nothing short of ligature above and below it would be of any avail in stopping the hæmorrhage. Had the wound been small, however, I question whether I should not have trusted either to pressure or to the lateral ligature, since I was strongly advised by several of my colleagues

<sup>1</sup> 'Lancet,' 1874, vol. ii, p. 544.

not to apply, if it could possibly be avoided, a circular ligature to the vein (on account of increasing the risk of gangrene); indeed, I hoped that on securing the artery the venous hæmorrhage might perhaps stop of its own accord, or at any rate be controlled by slight pressure over the wound. Langenbeck, as is well known, taught that ligature of the accompanying artery is often in itself sufficient to arrest hæmorrhage from a wound of the vein, and Dr. Lidell in his article on 'Injuries of Blood-vessels' in Ashhurst's 'International Encyclopædia of Surgery' quotes several cases in support of this view. The first surgeon to adopt the practice appears to be Gensoul,<sup>1</sup> who deliberately tied the femoral artery for the purpose of arresting hæmorrhage from the femoral vein, and similar successes are reported by Ward,<sup>2</sup> Cock,<sup>3</sup> and Halford.<sup>4</sup> The last-named surgeon further demonstrated that in the rabbit hæmorrhage from the femoral vein could be effectually arrested in this way. He placed a ligature loosely around the femoral artery, then opened the vein lower down, and found that the bleeding, which was profuse, immediately stopped on tightening the ligature. This experiment was repeated fifteen times with the same result. I have also myself seen several cases at St. Bartholomew's Hospital of accidental wound of the femoral vein while tying the femoral artery, in which the hæmorrhage ceased, or was quite easily controlled by pressure when the artery was occluded. In the case now brought before the Society, in which, however, the wound in the vein was large, and in the cases reported by Drs. Pilcher,<sup>5</sup> Gerster, Rose, and Kraske,<sup>6</sup> the ligature of the artery had no appreciable effect in controlling the hæmorrhage, and other means had to be resorted to. In

<sup>1</sup> 'Gaz. Méd. de Paris,' series 2, 1836.

<sup>2</sup> 'London Medical Gaz.,' 1845, N.S. 1, p. 987.

<sup>3</sup> 'Lancet,' 1853, vol. i, p. 296.

<sup>4</sup> 'Med. Times and Gaz.,' 1862, vol. i, p. 667.

<sup>5</sup> 'New York Med. Journ.,' 1884, p. 618.

<sup>6</sup> 'Centralblatt für Chirurgie,' 1880, p. 687.

eight cases collected by Mr. Pearce Gould<sup>1</sup> of wound of the femoral vein treated by compression there were six deaths, but as no details are furnished too much stress, as Mr. Gould himself says, should not be placed upon them in considering the effects of pressure in the treatment of a wound of this vessel. In the St. Bartholomew's Hospital cases referred to above, in which the vein was accidentally pricked in tying the femoral artery, no harm ensued when the ligature was withdrawn and the artery tied higher up. Where, however, the artery was tied at the same spot as that at which the vein was pricked, thrombosis of the vein and death from blood-poisoning I believe invariably followed. These cases, it should be mentioned, occurred at a time when the ligature round the artery was left hanging out of the wound, and before the days of antiseptic surgery. Arguing from these facts it would appear that there is some risk, to say the least, in a case of wound of the artery and vein to tie the artery above and below the wound and to leave the vein untouched, or to trust to pressure alone. If the wound of the soft parts could with absolute certainty be rendered and kept aseptic, the hole in the vein would probably, if small, heal up without further trouble, but when it is considered how likely the external wound is to suppurate, either from having been infected by the inflicting instrument or from the almost unavoidable bruising and tearing of the tissues by the extravasated blood, and the necessary manipulations in tying the artery, it does not seem that such practice can be generally recommended, and there is always a further danger, as mentioned by Mr. Gould, of the venous hæmorrhage, when apparently stayed, bursting out during some expiratory effort, an accident that happened to his patient.

<sup>1</sup> 'Lancet,' vol. i, 1887, p. 258.

5. *The question of the lateral versus the circular Ligature in wounds of large Veins.*

The lateral ligature, says Malgaigne,<sup>1</sup> is disavowed in theory and condemned in practice; Professor Gross<sup>2</sup> pronounces the lateral ligature highly dangerous, and considers it ought to be excluded from modern surgery. Dr. Lidell,<sup>3</sup> in his article on "Punctured Wounds of Veins" in 'Ashhurst's Encyclopædia,' after referring to a successful case, continues, "Nevertheless it (the lateral ligature) should not be imitated because of the great risk of secondary hæmorrhage." Mr. Pearce Gould, in his article in the 'Lancet,' says, "The relative value of the circular and lateral ligature is the same in the femoral as in other situations, and although many successful ligatures of large veins are recorded, the fatalities are too numerous to warrant its repetition except in the case of punctures and very small wounds." I do not know to what fatal cases Mr. Pearce Gould alludes, but Prof. Gross, Dr. Lidell, and Malgaigne apparently base their view of the dangers of the lateral ligature chiefly on a fatal case related by Travers, and on cases reported by Nélaton as occurring in the practice of Roux, cases, it may be remarked in passing, which happened at a time when suppuration in wounds was exceedingly common, and secondary hæmorrhage consequently far from rare. On the other hand, Messrs. Erichsen and Bryant both recommend that in small punctured wounds of large veins the lateral ligature should be applied, and I believe that several of my colleagues are in favour of it. The investigations of the subject here undertaken would lead me to the conclusion that the danger of lateral ligature has been over-estimated. I have collected sixteen cases of wounds of large veins thus treated (all I could find published), including those of

<sup>1</sup> 'Gaz. des Hôp.,' 1853, p. 88.

<sup>2</sup> 'American Journal Med. Science,' April, 1867, p. 318.

<sup>3</sup> Ashhurst's 'International Encyclopædia of Surgery.'



Travers and Roux. In thirteen cases the treatment was completely successful. In Guthrie's celebrated case, in which death occurred from exhaustion, the internal jugular vein, to which the lateral ligature had been applied, was found pervious at the post-mortem examination, and without a mark indicating the situation of the ligature. The four cases which have come under my own observation dispose me to regard lateral ligation in a favorable light. One of these was a wound of the subclavian in removing a tumour of the clavicle ; the three others were wounds of the internal jugular vein in excising a tumour in the neck. In one of the latter (my own case) the hæmorrhage was very profuse. The wall of the vein, however, was nipped up by forceps, a silk ligature applied, and the ends cut off. The wound healed without any trouble in a few days. The other three cases were also successful. Considering the risk of gangrene when the common femoral artery and vein are tied in their continuity before time has been allowed for the collateral circulation to become established, I should feel inclined, supposing the wound in the vein to be of moderate size, to apply the lateral ligature rather than the circular. But where the collateral circulation has become assured, and for wounds of the superficial femoral vein where the risks of gangrene are less, the circular ligature above and below the wound is probably the safer course, inasmuch as it is impossible in all cases of these injuries to be absolutely sure of obtaining healing of the soft tissues by the first intention.

### CONCLUSIONS.

The conclusions that I venture to draw, with some diffidence, from a consideration of my own case, and a review of the cases bearing upon it, are :

1. That when a large artery and vein, as the femoral, are involved in a punctured wound of the thigh, the safer course is to apply pressure in the way described above

for a few days to allow the collateral circulation to become established, and then to cut down and tie the proximal and distal extremities of both the artery and vein.

2. That immediate ligature (*i. e.* before the collateral channels have had time to enlarge) of both the femoral artery and vein, especially of the common femoral, for a wound of these vessels is liable to be attended with gangrene, although this risk is probably less than has been generally assumed.

3. That ligature of both vessels when in consequence of pressure, as of a tumour, the collateral circulation has become established, is attended with much less risk of gangrene.

4. That when the femoral artery and vein are wounded, ligature of the artery and pressure on the vein, if the wound is a mere puncture, is a safe treatment provided that the nature of the injury allows of reasonable prospects of the external wound being kept aseptic and uniting by the first intention.

5. That when the wound in the vein is too large to permit of treatment by pressure, the walls may be safely nipped up, and a ligature thrown around them without obliterating the calibre of the vessel; but that this procedure should only be resorted to, as in the former case, where there is a reasonable prospect of the wound healing by the first intention.

6. That, considering the grave risks of gangrene which attend the sudden obliteration of the common femoral vein, the lateral ligature should, in this situation, for all small and moderate-sized wounds which require immediate ligature, be the treatment adopted.

TABLE I.—Six Cases of Wound of the Femoral Artery and Vein. *Ligature not employed.*

No.	Reference.	Surgeon.	Sex, age.	Nature and situation of wound.	Vessels implicated.	Immediate treatment and results.	Later treatment; vessels ligatured.	Date of ligature after injury.	Remarks.	Death or re- covery.
1	Int. Encycl. Surg., iii, p. 176; Surg. Hist. War Rebel., vol. ii, part ii, p. 78 (H. Clinton)	—	M.	Pistol-shot, right thigh, lower third	Superficial femoral artery and vein severed	No treatment	—	—	Death in a few minutes	D.
2	Int. Encycl. Surg., p. 177; Surg. Hist. War Rebel., vol. ii, part ii, p. 85 (W. Neff)	—	M.	Gunshot wound, both thighs	Femoral artery and vein wounded	Tourniquet	Condition too serious for operation	—	Death in a few hours	D.
3	Surg. Hist. War Rebel., vol. ii, part iii, p. 13 (Bailey)	—	M.	Gunshot wound, thigh	Femoral artery and vein wounded	Expectant plan of treatment	No details given	—	Cause of death not stated	D.
4	Surg. Hist. War Rebel., vol. ii, part iii, p. 13 (Clarendon)	—	M.	Gunshot wound, thigh	Femoral artery and vein wounded	Expectant plan of treatment	No details given	—	Cause of death not stated	D.
5	Surg. Hist. War Rebel., vol. ii, part iii, p. 54, Case 38	Bliss	M. 30	Gunshot, right thigh	Femoral artery and vein wounded, profuse hæmorrhage	Compression ?	No details given	6th day	Amputation; pyæmia; death 27th day	D.
6	Surg. Hist. War Rebel., vol. ii, part iii, p. 55, Case 72	Burditt	—	Gunshot wound, left thigh	Femoral artery and vein severed	—	No details given	9th day	Amputation; death same day; shock and gan- grene	D.

TABLE II.—*Twelve Cases of Wound of the Femoral Artery and Vein. Immediate Ligature.*

No	Reference.	Surgeon.	Sex, age.	Nature and situation of wound.	Vessels implicated.	Immediate result.	Vessels ligatured.	Date of ligature.	Remarks.	Death or recovery.
1	St. Petersburg med. Wochen., 1884	Wahl	M. 20	Pistol-shot, lower third, right thigh	Superficial femoral artery and vein wounded	Arterio-venous murmur heard at bleeding spot	Superficial femoral artery and vein tied above and below wound	1 hour	Secondary hemorrhage 6th and 12th day; wound opened; proximal ends tied again	R.
2	Berl. klin. Wochens., 1881, Jan. 17, p. 38	Tillmans	M. 26	Punctured wound, knife, middle third thigh	Superficial femoral artery and vein wounded	Hæmorrhage into limb	Superficial femoral artery and vein tied	3 hours	—	R.
3	New York Med. Journ., 1884, Nov. 29th, p. 617	Pileher	M. 34	Punctured wound, knife, upper third thigh	Superficial femoral artery and vein wounded just below origin of profunda and long saphenous	Profuse hemorrhage	Superficial femoral artery and vein tied above and below wound	At once	No bad symptom; slight œdema when first began to use limb	R.
4	Lancet, 1878, vol. ii, p. 787	Goodall	M. 18	Punctured wound, thigh, 4½ in. below Poupert's ligament	Superficial femoral artery and vein severed	—	Superficial femoral artery and vein tied	At once	No œdema or other bad symptoms; wound healed in 8 weeks	R.

No.	Reference.	Surgeon.	Sex, age.	Nature and situation of wound.	Vessels implicated.	Immediate result.	Vessels ligatured.	Date of ligature.	Remarks.	Death or re- covery.
5	Volkmann's Sammlung klin. Vorträge, No. 29, 1875— 80, p. 754	Rose	M. 25	Punctured wound under Poupart's ligament, right thigh	Common femoral artery and vein wounded	Loud and rough blowing murmur	Common femoral artery and vein tied at site of wound	At once (22 hours)	Wound healed first intention; no oedema	R.
6	Lancet, 1883, vol. i, p. 106	Clement Lucas	M. 35	Incised wound of thigh, and middle third	Superficial femoral artery and vein, and long sapherous nerve divided	—	Superficial femoral artery twisted; vein tied	At once	<i>Gangrene</i> 5th day; amputation 9th day at seat of injury	R.
7	Centralblatt, 1880, 43, Oct. 23rd, p. 689	Volkmann (reported by Kraske)	M. 21	Shot, upper third, right thigh, in duel	Common femoral artery and vein wounded be- low Poupart's ligament	Profuse hæmor- rhage	Common femoral artery tied; also vein as hæmorrhage did not stop	At once	Limb became cold and paralysed; superficial veins dilated; <i>gangrene</i> 2nd day	D. 11th day.
8	Beitrag zur Kenntniss der Aneurisms, Diss. Zurich, 1881	Rose (reported by Bloch)	M. 22	Stab with knife, right thigh, just below Poupart's ligament	Femoral artery and vein wounded	Tumour formed immediately without pulsa- tion; no pulse in artery below	Femoral artery and vein tied 1½ centimetres below origin of profunda	At once (2 hours)	<i>Gangrene</i> ; delirium (chronic drinker)	D. 7th day; throm- bosis of femoral vein.
9	Beitrag zur Kenntniss der Aneurisms, Diss. Zurich, 1881	Rose (reported by Bloch)	M. 53	Stab with knife, middle third, left thigh	Superficial femoral artery severed; vein wounded	Leg livid and swollen	Femoral artery and vein tied just above ad- ductor magnus opening	5 hours	Delirium (a heavy drinker); loss of sensation in leg; no bleeding on puncture	D. 4th day; clots in femoral vein.

10	International Encyclopædia of Surgery, vol. iii, p. 177 (Gross)	Gross	M.	Gunshot wound, middle third, thigh	Superficial femoral artery severed	Hæmorrhage stopped by finger	Superficial femoral artery and vein tied	24 hours	4th day, swelling and œdema; 5th day, secondary hæmorrhage arrested by pres- sure; 8th day, <i>gangrene</i> ; 9th, death. Distal liga- ture had slipped; muscles infiltrated with pus	D.
11	Surg. Hist. War Rebel., part iii, vol. ii, p. 49, Case 107 (South)	Dougherty	M.	Gunshot wound, right thigh	Femoral artery and vein	—	Femoral artery and vein tied	Same day	Amputation of thigh; <i>presumably</i> <i>gangrene</i> ?	D. 30th day.
12	Beitrag zur Kenntniss der Aneurisms, Diss. Zurich, 1881	Rose (reported by Bloch)	M. 27	Stab by knife, right thigh, upper third	Profunda vein and artery; hæmorrhage	Femoral artery tied; hæmor- rhage continued	Profunda artery and vein; also superficial femoral artery and vein	Soon (a few hours)	No disposition to <i>gangrene</i> ; ex- tremity continued warm and pulse- less; no nervous disturbance	R.

TABLE III.—*Twenty Cases of Wound of Femoral Artery and Vein. Treated first by Pressure, afterwards by Ligature of both Artery and Vein.*

No.	Reference.	Surgeon.	Sex, age.	Nature and situation of wound.	Vessels implicated.	Immediate treatment and results.	Vessels ligatured.	Date of ligature after injury.	Remarks.	Death or recovery.
1	Unpublished	Walsham	M. 19	Punctured wound, middle third, right thigh	Superficial femoral artery and vein	Pressure; wound healed; arterio-venous aneurism formed	Superficial femoral artery and vein in Hunter's canal	4 days	No bad symptom	R.
2	Brit. Med. Journ., 1875, i, p. 713	Czerney	M. 20	Knife stab, lower third, thigh	Superficial femoral artery and vein	Instrumental and digital compression; arterio-venous aneurism formed	Superficial femoral artery and vein	8 weeks	—	R.
3	Lancet, 1875, i, p. 568	Annandale	M. 10	Wound with scissors, popliteal space	Popliteal artery and vein	Pressure and bandages; arterio-venous aneurism formed in 3 days	Popliteal artery and vein tied	3 mos.	—	R.
4	Berlin. klin. Woch., 1883, 2, p. 19	Ebenau	M. 21	Punctured wound; shot, middle third, right thigh; profuse hæmorrhage	Superficial femoral artery and vein	Pressure; elastic bandages; arterio-venous aneurism formed in a week	Superficial femoral artery and vein in Hunter's canal	3½ mos.	—	R.
5	St. Petersburg med. Woch., 1883, No. 7, p. 53	Wahl	M. 23	Punctured wound, middle third, left thigh	Superficial femoral artery and vein	Pressure by bandage; arterio-venous aneurism soon formed	Superficial femoral artery and vein	12 years	Great swelling of veins for 5 years before ligature	R.

6	Med. and Surg. Hist. War of Rebel., vol. ii, pt. iii, Case 20, p. 47 (Coble)	—	M. 20	Gunshot wound, left thigh	Superficial femoral artery and vein	Pressure; diffuse femoral aneurism formed	Superficial femoral artery and vein	7 weeks	—	R.
7	Lancet, 1874, vol. ii, p. 546	Hamilton (Richmond Hospital)	M.	Stab, thigh	Superficial femoral artery and vein	Compression; arterio-venous aneurism formed	Superficial femoral artery tied above and below wound; vein also tied (?)	2 mos.	—	R.
8	St. Barth. Hosp. Reports, vol. iii, p. 448	Coote	M. 49	Punctured wound, right thigh	Superficial femoral artery and vein	Pressure; arterio-venous aneurism formed; afterwards instrumental pressure; consolidation	Superficial femoral artery and vein	3 mos. 17 days after injury; 1 month after pressure	Aneurism was consolidated; injudicious examination; pulsation recommenced, then ligature and opening of sac; secondary hemorrhage; amputation and death	D.
9	Guy's Hosp. Reports, ser. ii, vol. viii, p. 228	Bransby Cooper	M. 15	Wound of thigh with penknife	Superficial femoral artery and vein	Pressure; arterio-venous aneurism formed	Superficial femoral artery and vein	10 days	No tendency to gangrene; no fall of temperature; no cedema; pyæmia; femoral vein had not been entirely included in the ligature	D.
10	Bull. et Mém., 1883, t. ix, p. 285	Nélaton	—	Gunshot wound, right thigh	Superficial femoral artery and vein	Compression; arterio-venous aneurism formed in a few days	Superficial femoral artery and vein tied above and below sac; sac opened; plug of charpie in wound	1½ mos.	Ligature separated 8th day, septic suppuration; 12th, secondary hemorrhage; cauterization and perchloride of iron; repeated hemorrhage	D.



No.	Reference.	Surgeon.	Sex, age.	Nature and situation of wound.	Vessels implicated.	Immediate treatment and results.	Vessels ligatured.	Date of ligature after injury.	Remarks.	Death or re- covery.
11	Bull. et Mém., 1883, April, p. 264	Verneuil	M. 17	Punctured wound, popliteal space	Popliteal artery and vein	Pressure; wound healed quickly; arterio- venous aneurism formed (aneu- rismal varix)	Compression di- gital and direct; popliteal artery and vein tied; sac opened; four muscular veins tied also; hæmorrhage continued; all tissues tied between artery and bone	3 years	No gangrene; secondary hæmor- rhage 23rd day; cautery	R. Slight oedema and dilated veins.
12	Surg. Hist. War Rebel., part iii, vol. ii, p. 45; also in Int. Encycl. Surg., vol. ii, p. 213 (Rapp)	Thompson	M. 20	Gunshot wound, left thigh	Superficial femoral artery and vein wounded	Profuse hæmor- rhage; arterio- venous aneurism formed; 9th day hæmorrhage from wound	Ligature of superficial femoral artery and vein	9th day	Gangrene 12th day (3rd day after ligature); pro- funda vein plugged; profunda artery not enlarged	D. next day.
13	Med. Times and Gaz., 1860, vol. i, p. 216	Hilton	M.	Gunshot wound, right thigh, 3 in. below Poupart's ligament	Wound of superficial femoral artery; vein seen full of flowing blood	Ligature of artery; hæmor- rhage 7 days after from vein	Ligature of vein	7th day	Gangrene com- mencing, due to clamp which was placed on artery slipping and com- pressing vein above profunda	D. 13th day.
14	Surg. Hist. War Rebel., vol. ii, part iii, p. 48, Case 59	Pineo	M.	Gunshot wound, right thigh, May 25	Wound of femoral artery and vein	Not stated; no details	Femoral artery tied both ends; vein included in ligature	June 12th, 17th day?	Cause of death not stated	D. next day.

15	Surg. Hist. War Rebel., vol. ii, part iii, p. 48, Case 65	Norris	M. 18	Gunshot wound, right thigh, April 6	Femoral vein ruptured	Not stated; no details	Femoral artery tied at middle portion; vein above and below rupture	April 25th, 19th day?	Recurrent hæmorrhage, May 5th (10th day after ligature)	D. May 7th.
16	Surg. Hist. War Rebel., vol. ii, part iii, p. 47, Case 44	—	M.	Gunshot wound, right thigh, July 17th	Wound of femoral artery and vein	Not stated; no details	Femoral artery tied at proximal end; vein proximal and distal end	July 23rd, 6th day	Gangrene	D. July 27th.
17	Lancet, 1853, vol. i, p. 296	Adams and Ward	M. 20	Punctured wound, middle third, left thigh; considerable hæmorrhage	Superficial femoral artery and vein, also profunda artery	Graduated compress; secondary hæmorrhage 9th day; wound sloughy	Superficial femoral artery tied; vein divided transversely to allow it to collapse; profunda artery also tied	9th day	Gangrene 6th day. Saphenous vein found plugged	D. 11th day.
18	Deuts. Zeits. f. Chir., 1875, B. v, p. 174 (Rabe's cases)	Langenbeck	M. 20	Punctured wound with knife	Superficial artery and vein	Pressure? arterio-venous aneurism formed	Superficial femoral artery and vein	12th day	No gangrene, but œdema. Death probably due to pulmonary embolism	D. 7th day.
19	Pennsylv. Hosp. Rep., i; Deuts. Zeits. f. Chir., 1875, B. v, p. 175 (Rabe's cases)	Morton	M. 21	Punctured wound	Superficial femoral artery and vein	Pressure? arterio-venous aneurism formed	Superficial femoral artery and vein	8th day	No bad symptoms	R.
20	Chir. Klin., Wien, 1868, p. 158	Billroth	M. 19	Punctured wound, upper third, left thigh	Superficial femoral artery and vein	Pressure for six weeks by a barber; arterio-venous aneurism formed	Superficial femoral artery and vein	6 weeks	Secondary hæmorrhage; ligature of common femoral, then <i>gangrene</i>	D.

TABLE IV.—*Four Cases in which the Femoral Artery and Vein were Ligatured for Wound inflicted during the Removal of a Tumour.*

No.	Reference.	Surgeon.	Sex, age.	Cause.	Vessels implicated.	Ligature.	Remarks.	Death or re- covery.
1	Handb., B. ii, p. 989 ; also Berl. klin. Woch., 1881, Jan. 24, p. 55	Stromeyer	—	Removal of tumour, anterior crural nerve	Piece of femoral artery and vein removed with tumour	Artery and vein tied above and below wound	No gangrene	R.
2	Beiträge zur Chirurgie, Volkman, p. 249	Volkman	F. 46	Removal of myxoma, left thigh, as big as child's head	Piece of femoral artery and vein below origin of profunda removed with tumour	Artery and vein tied above and below wound	No gangrene	R.
3	Int. Encycl. Surg., vol. iii, p. 213 ; also Berl. klin. Woch., Jan. 24, 1881	v.Oettingen	—	Removal of tumour of thigh	Wound of common femoral vein during operation	Double ligature of common femoral vein ; leg became cyanotic ; artery then tied ; cyanosis disap- peared	No sign of gangrene ; septicæmia, fatal on 5th day	D.
4	Chir. Klin., Wien, 1867-70, p. 325 (Billroth) ; also Berl. klin. Woch., 1881, Jan. 24, p. 56	Billroth	M.	Removal of cancerous glands from thigh	Wound of common femoral vessels	Common femoral artery and vein tied	No sign of gangrene ; septicæmia, fatal on 2nd day	D.

TABLE V.—*Sixteen Cases of Wound of Femoral Artery and Vein. Treated by Pressure. Followed by Formation of Arterio-venous Aneurism.*

No.	Reference.	Surgeon.	Sex, age.	Nature and situation of wound.	Vessels implicated.	Immediate treatment and results.	Later treatment.	Date of later treatment.	Remarks.	Death or recovery.
1	Brit. Med. Journ., vol. i, 1887, p. 1271	Godwin	M. 29	Punctured wound, bamboo stick, right thigh, 1½ in. above patella; penetrated 2 in.	Superficial femoral artery and vein wounded; much extravasation	Pressure; wound healed in 14 days; arterio-venous aneurism formed	Digital pressure	—	No alteration in aneurism	R.
2	St. Barth. Hosp. Rep., vol. xii, p. 157	Smith	M. 14	Punctured wound, middle thigh, pen-knife	Superficial femoral artery and vein wounded	Pressure; wound healed in few days; aneurismal varix formed	Pressure	21 days	Venous murmur heard still	R.
3	Clin. Soc. Trans., vol. viii, p. 175	Hulke	M. 44	Explosion of pistol, wound upper third thigh	Superficial femoral artery and vein	Pressure; wound healed in 2 days; pain and throbbing; aneurismal varix formed	Pressure, direct and indirect	—	Discharged wearing elastic stocking; aneurism not improved	R.
4	Med. Times and Gaz., 1888, vol. ii	Pick	M. 28	Pistol shot, right thigh, upper third	Superficial femoral artery and vein	Wound drained and bandaged; healed in a month; aneurismal varix formed	Pressure and elastic bandage	1 month about	Aneurism continued	R.
5	L'Union Méd., 1884, ser. 3, vol. xxxvii, p. 973	Schwartz	M. 18	Punctured wound of thigh, much hæmorrhage	Superficial femoral artery and vein	Pressure; 2ndry hæmorrhage; compression again; arterio-venous aneurism formed	Compression, elastic bandage, Esmarch's bandage	9 mos.	Aneurism not improved or very slightly	R.

No.	Reference.	Surgeon.	Sex, age	Nature and situation of wound.	Vessels implicated.	Immediate treatment and results.	Later treatment.	Date of later treatment.	Remarks.	Death or recovery.
6	Bull. et Mém. Soc. de Chir. de Paris, 1884, vol. x, p. 332	Trelat	M. 15	Punctured wound with knife middle left thigh; profuse hæmorrhage.	Superficial femoral artery and vein	Compression 15 days; wound healed; arterio-venous aneurism formed	Elastic bandage, direct compression, indirect pressure; arterio-venous converted into ordinary aneurism	About a month	Arterial aneurism cured by continued pressure. Trelat remarks the wound in vessels was probably small, hence the success	Cured.
7	Progrès Méd., No. 30, 1892, p. 582	Caraff	M. 32	Gunshot wound, thigh, upper third	Superficial femoral artery and vein	Compression 2 days; arterio-venous aneurism formed next day; digital, shot-bag	Digital compression; instrumental continued; shot-bag for 12 days	—	Phlegmon; incisions; sepsis; enormous abscess	D. 7 weeks.
8	Edin. Med. Journ., July, 1869	Spence	M. 15	Wound, upper third thigh	Femoral artery and vein	Pressure; wound healed 10 days; arterio-venous aneurism formed after walk	Ligature of the artery above and below sac	1 month and 4 days	—	B.
9	Gaz. Méd. de Paris, 1896, vol. iii, p. 265	Polailon	28	Punctured wound, upper third right thigh; profuse hæmorrhage	Superficial (?) femoral artery and vein	Compression for 4 months; wound long in healing; left the hospital with arterio-venous aneurism (aneurismal varix)	Compression; electro-puncture; septicæmia	7 years	Bruit prevented sleep	D.
10	Int. Encycl. Surg., vol. iii, p. 247	Cutter	M. 16	Wound, inner part left thigh, 2 in. below Poupart's ligament; penknife	Femoral artery and vein; profuse hæmorrhage	Pressure; wound healed in a week; arterio-venous aneurism formed 8 years after from fatigue	External iliac tied; still later common iliac	8 years	Peritonitis; aneurismal varix at post-mortem	D.

11	Hennen's Military Surgery, 3rd edit., p. 185	Hennen	M. 24	Wound with musket ball	Wound of femoral artery and vein	Pressure; wound healed 61 days; arterio-venous aneurism began to form in 3 weeks	tied			
12	Gaz. Méd., 1841, p. 172	Lallemand	M. 27	Punctured wound, middle third thigh, profuse hæ- morrhage	Wound of femoral artery and vein	Pressure; wound healed; arterio- venous aneurism soon formed	Femoral artery tied, then external iliac	5 years	Recurrent hæmor- rhage	D.
13	Amer. Journ. Med. Sci., 1838, xxii, 329	Morrison	—	Stab in thigh	—	—	External iliac tied	—	—	D.
14	Gaz. Hebd., 1864, tome i, p. 166	Gayet	M. 19	Punctured wound, upper third right thigh	Common femoral artery and vein sup- posed to be wounded	Pressure 5 days; digital pressure; secondary hæmorrhage; arterio-venous aneurism formed	External iliac tied	23 days	Peritonitis; septi- cæmia; profunda artery and vein found wounded; femorals found intact	D. 10 days.
15	Med.-Chir. Trans., 1851, p. 327	Cock	M. 17	Punctured wound, lower third thigh	Popliteal artery and vein	Carefully ap- plied pressure 12 days; secondary hæmorrhage; ligature of femoral artery; wounds healed; arterio-venous aneurism formed	Incision into abscess, formed in connection with aneurism; profuse hæmor- rhage	11 years	Amputation	R.
16	Med. Times and Gaz., July 27, 1867, p. 87	Beaumont	M. 45	Punctured wound, pocket- knife; pro- fuse hæmor- rhage	Superficial femoral artery and vein	Pressure not stated; wound healed in a few days; arterio- venous aneurism formed	Died under chloroform pre- paratory to ligature of external iliac	10½ years	Aneurism gave no trouble for 10 years; then en- larged suddenly from strain in riding	D.

TABLE VI.—*Sixteen Cases of Wounds of Large Veins treated by the Lateral Ligature. Successful 13. Unsuccessful 3.*

No.	Reference.	Surgeon.	Sex, age.	Cause.	Vessels implicated.	Ligature.	Remarks.	Death or recovery.
1	Cornack's Monthly Journ. Med. Sciences, 1844, p. 783	Watman, 1823	—	Removal of tumour of neck	Longitudinal opening of 2½ lines in internal jugular vein	Lateral ligature	Ligature separated 12th day	R.
2	Legouest's Traité de Chirurgie d'Armée, 1863, p. 429	Bégin	—	Removal of tumour of neck	Wound of internal jugular vein	Lateral ligature	—	R.
3	Metropolitan Hospital (unpublished)	Walsbam	—	Removal of cancerous glands	Puncture of internal jugular vein; profuse venous hemorrhage	Coats around wound nipped up, and lateral ligature applied; ends of ligature cut short	No further trouble	R.
4	St. Barth. Hospital (unpublished)	Callender	—	Removal of tumour of neck	Wound of internal jugular vein	Lateral ligature	No bad symptom	R.
5	Ashhurst's Int. Encycl. Surg., p. 199	Lidell	—	Dissecting out deep-seated tumour of neck	Puncture of internal jugular vein	Lateral ligature	—	R.
6	St. Barth. Hospital (unpublished)	Langton	—	Removal of tumour of neck	Wound of internal jugular vein	Lateral ligature	—	R.
7	Int. Encycl. Surg. (Dr. Lidell)	McClellan	—	Removal of tumour of groin	Wound of internal saphenous vein at its entrance to femoral	Lateral ligature	—	R.

	Bull. et Mém., 1871, Oct. 18th	Despres	—	Gunshot wound, middle third thigh	Suppuration and secondary hæmor- rhage from femoral vein	Lateral ligature	Separated 6th day	R.
8			—					
9	Malgaigne's Anatomy, p. 342	Blandin	—	Removal of can- cerous glands	Axillary vein wounded	Lateral ligature	—	R.
10	Malgaigne's Anatomy, p. 342	Bérard	—	Not stated	—	Lateral ligature	—	R.
11	Malgaigne's Anatomy, p. 342	Richet	—	Not stated	Femoral vein	Lateral ligature	Cicatrization of wound in 8 days	R.
12	Gazette des Hôpitaux, 1858, p. 88	Roux	—	Operation on neck	Internal jugular vein	Lateral ligature	Secondary hæmorrhage on separation of ligature	D.
13	Gazette des Hôpitaux, 1858, p. 88	Roux	—	Operation on neck	Internal jugular vein	Lateral ligature	Secondary hæmorrhage	D.
14	St. Barth. Hospital (unpublished)	T. Smith	—	Removal of clavicle for sar- coma	Punctured wound of subclavian vein	Lateral ligature	Recovery without a bad symptom	R.
15	Malgaigne's Anatomy, p. 344	Travers, 1816	—	Tying femoral artery	Punctured wound of femoral vein	Lateral ligature	Secondary hæmorrhage on 12th day, at sepa- ration of ligature	D. Ex- haus- tion.
16	Diseases and Injuries of the Arteries, 1880, p. 328	Guthrie	—	Cut throat	Wound of internal jugular vein ; carotid also injured	Lateral ligature	The internal jugular vein was found pervious, and without a mark indicating the seat of previous wound and ligature	D. Ex- haus- tion.



(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 361.)

A CASE  
OF  
DOUBLE NEPHROLITHOTOMY  
IN WHICH LATERAL AND MEDIAN LITHOTOMY  
HAD BEEN PREVIOUSLY PERFORMED,  
WITH REMARKS ON SYMPATHY BETWEEN THE KIDNEYS.

BY  
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As a contribution to renal surgery I trust that the following case will be thought worthy of the attention of the Society. The history is a long one, but I will endeavour to tell it without excess of words.

W. W—, æt. 22, one of five, all healthy, a doubler in a tin mine in South Wales, an occupation involving much stooping and lifting heavy weights, was sent to me at St. Mary's Hospital by Dr. Evans, of Llanelly, on November 26th, 1886. For the last eight or ten years he had had pain in the region of the left kidney, but there was nothing specially noticeable about his urine until four years ago, when it was often high coloured and rich in sediment, and occasionally he passed gravel. In the course of a year from this time he began to have sudden stoppage in the act of micturition, with bladder irritability,

and at times the pain in his loin was more severe, and showed all the characteristics of transient renal colic. The evidence of stone in his bladder became more and more decided, and fifteen months ago, by lateral lithotomy at his home in Wales, three calculi were removed, the largest of them being the size of a hazel-nut, of a fawn colour and smooth surface. In a fortnight he was well, but on the day after the wound was closed he was seized with pain, much more violent than ever before, in the left loin; he was conscious of a stone passing down the ureter, and on the following day there was every indication of another stone being in his bladder.

At this time there was some blood in his urine, and probably pus also, for he describes his water as having deposited a thick white sediment. Since this calculus descended into his bladder fifteen months ago, he has never long been free from the sensation of its presence, though he speaks of the bladder pain as having been somewhat different from that caused by the first set of stones, the stone feeling to him as if it lodged at times in some pouch near the neck of the bladder. The pain is often very acute at the close of micturition, and compels him to keep at absolute rest on his back for three or four hours. He has moreover a continuous dull aching pain in his left loin, which is made acute by lying on his right side. He can precisely indicate the seat of pain, about midway between the last rib and the anterior superior spine of the ilium, and although not actually tender, he very much dislikes pressure at this point. No tumour is to be felt. It is some time since he had an attack of severe renal pain, and he does not think that any stone has descended into his bladder since the one now in it fifteen months ago. His general health has very much deteriorated during the past year; he is in constant pain, his sleep is bad, he has lost flesh, and is quite unfit for work. His urine occasionally contains a small quantity of blood, is acid, of a sp. gr. varying from 1010 to 1020, with numerous pus-cells, but no casts. A small stone in the bladder

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was detected by sounding on November 29th, and from his previous history and present symptoms the further diagnosis was made that there was in all probability pyelonephritis with calculi on the left side. Nothing pointed to any mischief in the right kidney.

The question now arose as to the best course to be adopted for the removal of the stones, and it was decided first of all to take the calculus from the bladder, and then, should pain in the left loin continue in its former intensity, diminishing thereby the likelihood of its being a reflex pain from bladder disturbance, to perform lumbar nephrolithotomy. The stone in the bladder being small, and there being obviously some considerable degree of cystitis, I determined to extract it by median perineal incision, with the object of draining the bladder freely afterwards and keeping it at perfect rest. I deemed it better in the circumstances not to use the lithotrite, for fear of setting up more cystitis, which might possibly affect the kidney injuriously. Accordingly, on December 4th, a small calculus weighing forty-five grains (and composed in the main of oxalate of calcium with traces of phosphate of lime and magnesium as determined for me by Mr. Thompson) was removed from the bladder. The lithotomy wound calls for no comment. By December 12th he was passing water naturally *per urethram*, his temperature was normal, and in himself he felt better, having decidedly less pain in the back, and being free at any rate from one source of anxiety. By December 17th, the perineal wound was quite healed.

On December 12th, eight days after the lithotomy, he passed a considerable quantity of gravel, but had no acute pain. Early on the morning of the 13th, however, he began to feel more aching in the loin. This increased gradually during the day, and culminated in the afternoon in very violent renal pain, and his temperature rose to 101.8° F. This attack seemed to settle the question as to there being stones in his left kidney or its pelvis, and accordingly, on December 15th, this kidney was explored by

the usual transverse incision in the loin. There was considerable difficulty in making out where the kidney lay. It could neither be seen nor felt, but there came into view a large cyst, which looked like, and at first was thought to be, distended colon. The real nature of it having at length been determined by puncture with a hypodermic syringe, the cyst was freely incised, and exit was given to some ten ounces of offensive urinous pus, and with it two small calculi, each of them having a highly polished surface, with distinct facets, and about half the size of that which had been previously taken from the bladder. (These also are mainly composed of oxalate of calcium.) The kidney could now be felt high up under the ribs, and evidently very shrunken and small. No calculus could be detected in it, nor surface irregularity, but its extreme wasting led more than one of my colleagues to suggest that it would be better at once to remove it, as being a useless organ. I was not myself of that opinion, and the sequel of the case will show that we did wisely to leave it where it was. It helped very largely, I think, on a future day to save the patient's life.

For four or five days after the operation the patient had a great deal of pain in the neighbourhood of the wound, and opium had to be frequently given to him. There was, however, nothing wrong to be noticed about the parts, and urine escaped freely from the side. There was in fact every indication of the functional activity of this kidney, while the record of the urine passed by the urethra shows that the right kidney was in all probability excreting some twenty to thirty or more ounces a day.

The drainage-tube was not finally removed until January 27th, for it had been deemed advisable to keep it in thus long in order that the cyst might be irrigated and freely drained. The temperature had been normal after January 10th, and so it continued until February 18th, by which date the wound was soundly closed. We had been disappointed, however, to note that the pus had not really

been diminishing in quantity, and notwithstanding his general wellbeing and great improvement in appearance, and his entire freedom from pain or discomfort on the left side, he more than once complained, in the latter half of January, of feeling pain for a few moments in the region of the right kidney. I tried to calm his anxiety and our own by the suggestion that this pain was due to a distended colon, but its more frequent recurrence in the early half of February, and the decided increase in the quantity of pus, made me very suspicious, and I more than once remarked that the right kidney in all probability was affected as the left had been.

All doubt was set at rest on this point on February 18th by rise of his temperature to  $101^{\circ}$  F., simultaneously with a very sharp attack of pain in the right loin. On the following day he was much worse; his temperature was  $103^{\circ}$  F., he had several rigors, incessant vomiting, retraction of the right testis, intense pain, and all the symptoms were present of a very severe attack of renal colic. It was noted that on February 18th the quantity of urine suddenly dropped from forty-two to twenty ounces, and both on this and the two following days there was a great diminution likewise in the quantity of pus. On the 19th the urine had again risen to its former volume of forty ounces, and on the 22nd the pain and other symptoms of colic were very much less, and simultaneously there was a vast increase in the amount of the pus, far exceeding any previous quantity. Rightly or not these phenomena were interpreted as a sign that there had been obstruction somewhere to the flow of urine and pus from the right kidney, and that the left kidney, having its pelvis now pretty well free from suppuration, was really the source of the urine which reached the bladder. Now it was that the left kidney was in all probability the means of saving the patient from grave and imminent danger. The right kidney had either struck work, or had been mechanically prevented from excreting urine, and it seems not unreasonable to think that its chances of recovery

might have been worse, or even annihilated altogether, had there been no kidney on the opposite side.

The 24th and 25th of February brought a return of the renal pain, and this was now accompanied with much local tenderness and some suspicion of resistance and fullness in the loin. These were so marked on the 25th that it was decided, more especially as the man had had another very severe rigor, to delay no longer; and accordingly on this day the right kidney was exposed by transverse incision in the loin. It was found to be surrounded by stinking urinous pus, and the finger passed easily through an opening into the pelvis. It was evident that a collection of pus in the pelvis had burst into the perinephral tissue, and so had set up general suppuration around the gland. The kidney itself was considerably hypertrophied, but its appearance and feel, as far as feel could tell, were perfectly healthy. I could find no stones. There was much collapse after the operation, but otherwise the patient did well. The temperature fell at once to normal, urine flowed freely from the side for a fortnight, and then began to diminish, yet it did not entirely cease until March 18th. The wound was rather slow in healing and was not finally closed and sound until the 8th of May. He had all along been very much improving, was free from pain and gaining flesh, and expressed himself as more comfortable than he had been for years. The quantity of urine had steadily increased, and March, April, and May saw it ranging from forty to sixty ounces in the day. It still contained some pus, and it should be noted that once, and once only, two days after the second operation, he said he felt sure that another stone was in his bladder. He had, however, already gone through so much that I felt loath to sound or otherwise disturb him, hoping that if there were a calculus it might ultimately be expelled. He left the hospital on May 26th, well in himself, but still passing a very small quantity of pus, and still complaining of some pain in both loins, which was attributed to stretching of cicatricial

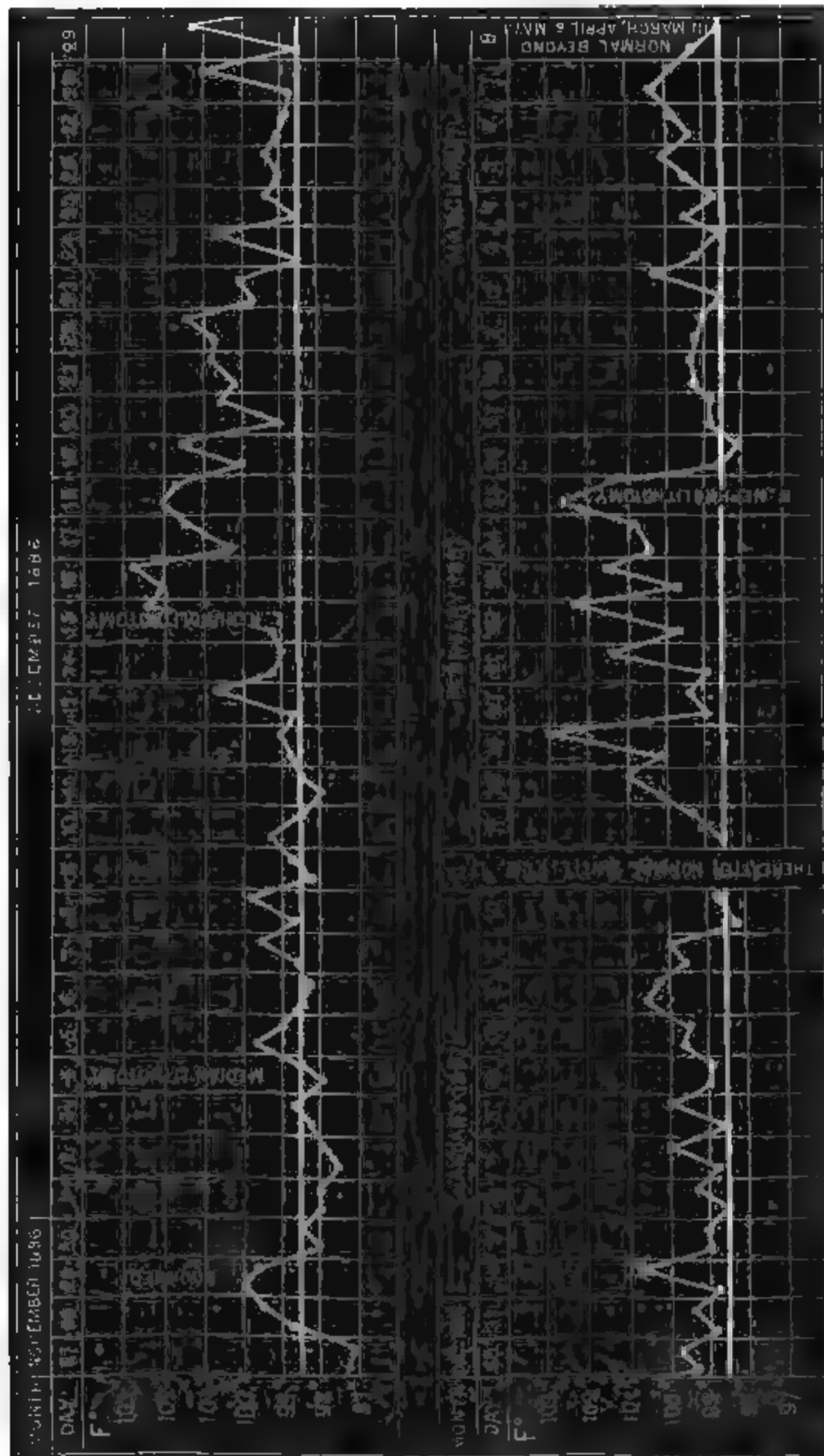
tissue at the site of the wounds. I have heard of him often since, and saw him last on October 24th. He then told me that a week after leaving the hospital he passed four very small stones, after a slight attack of right-sided pain. These in all probability were the stones which, being very small and weighing in all not more than twenty grains, were missed at the second operation, and which had been dislodged from a position of rest, either in the pelvis of the kidney, or more probably the bladder, by his journey home. With this exception he had been free from pain and had gained steadily in weight, health, and strength. He can hold his water for the natural length of time, and only very seldom is there anything which looks like pus. The cicatrices are sound, and there is no tenderness even on somewhat rough manipulation. In January again he wrote that he was better than he had been for years.

*Table of Urine passed in 24 hours.*

Date. 1886.	Ounces.				Total in 24 hours.	Date. 1887.	Ounces				Total in 24 hours.		
	A.M.		P.M.				A.M.		P.M.				
Dec. 5	...	18	...	11	...	29	30	...	30	...	—	...	—
6	...	14	...	10	...	24	31	...	28	...	—	...	—
7	...	16	...	13	...	29	1887.						
8	...	15	...	14	...	29	Jan. 1	...	28	...	24	...	52
9	...	18	...	—	...	—	2	...	—	...	22	...	—
14	...	23	...	—	...	—	3	...	36	...	35	...	71
15	...	Operation.					4	...	—	...	28	...	—
16	...	13	...	10	...	23	5	...	48	...	36	...	84
17	...	14	...	8	...	22	6	...	40	...	22	...	60
18	...	16	...	12	...	28	7	...	44	...	22	...	66
19	...	16	...	22	...	38	8	...	28	...	24	...	52
20	...	20	...	16	...	36	9	...	30	...	28	...	58
21	...	20	...	24	...	44	10	...	14	...	16	...	30
22	...	18	...	22	...	40	11	...	12	...	16	...	28
23	...	20	...	21	...	41	12	...	16	...	16	...	32
24	...	18	...	20	...	38	13	...	18	...	20	...	38
25	...	20	...	32	...	52	14	...	24	...	24	...	48
26	...	15	...	17	...	32	15	...	28	...	20	...	48
27	...	24	...	40	...	64	16	...	23	...	24	...	47
28	...	26	...	28	...	54	17	...	26	...	22	...	48
29	...	36	...	24	...	60	18	...	30	...	18	...	48







Beyond the record which has been already given there are one or two points of interest in this case to which I should now like to refer. It will be remembered that in the last session of the Society the discussion upon a "Case of Obstruction of one Ureter by a Calculus, accompanied by complete Suppression of Urine," brought forward by Mr. Godlee, was directed to the supposed "sympathy" between the two kidneys. The chief objection raised to the state of the one kidney having actually caused suppression of urine in the other, by sympathy, was in that case the fact that the other and presumably uninjured kidney was not a perfectly healthy organ ('Proc.,' N. S., No. 16, p. 238), that it had in reality been diseased for some time before the fatal attack seized the patient. Mr. Godlee had no doubt that a diseased would be more disposed than a healthy kidney to stop its secretion under the influence of irritation on the opposite side, not meaning to say, present knowledge perhaps not letting him go as far as to say, that such irritation would give rise to suppression in a perfectly healthy gland. With that opinion I should feel inclined to agree, but I would ask if this apparent sympathy may not rather be due to the irritation in one kidney "finding out," as it were, the mischief which is going on, and may have been going on for some time unsuspected or unknown in the fellow organ. Constitutional crises of very different kinds may exercise a like influence, and the "finding out" of a kidney's weakness may be the work of irritation or disturbance in other parts than the opposite kidney. This was what happened, I take it, in a case at this moment (January, 1888) under my care, the case of a patient from whose left kidney a large calculus was removed six weeks ago, and who it is believed has a calculus to be presently taken from her right kidney also, and in whom the first symptom of any kidney trouble whatever arose a fortnight after the operation of hysterectomy by my late colleague Dr. Meadows early last year. The symptoms began with great acuteness and suddenness,

with high temperature, violent pain, and frequent vomiting, with rigors and pus in the urine, which rapidly increased in quantity. It was believed at the time, and with this diagnosis already made she first came under my observation, that an acute suppurative pyelitis had been set up by direct extension from the hysterectomy wound, a wound which nevertheless had healed without untoward symptoms. I operated accordingly in the expectation of having to drain a suppurating pelvis, and possibly abscess cavities in the kidney itself. But the kidney was healthy, and a calculus firmly lodged in the hilum was the only thing wrong. The stone must have been there before Dr. Meadows removed her uterus, but its existence was first made manifest by the constitutional disturbance which his operation entailed. We may conjecture—we cannot of course say with certainty—that irritation in one kidney really does no more than this to its fellow, and there is no closer bond of sympathy between them.

Again, a renal calculus may be long quiescent, and give rise to no symptoms, neither pain nor pus, and the kidney in which it lies may excrete a normal quantity of perfectly healthy urine, yet nevertheless the kidney so affected cannot be as certainly healthy as one in which there is no stone. The gland is predisposed by the very presence of the stone to suffer, and it may even be to fail in conditions of constitutional disturbance, in conditions such as may arise, for example, from operation upon its fellow, and by the stress then thrown upon it its own weakness is revealed. A goodly number of cases are on record in which this apparently has been the sequence of events, while in none of them can it be said that the disturbance in the one kidney directly caused the mischief in the other. Thus in a case of Dr. F. Lange's (given in detail in Brodeur's work, '*Affections du Rein*,' 1886, p. 320) nephrolithomy was performed on the left side, where for years there had been pain, on October 2nd, and all went well until November 25th, when the patient was seized with severe pain, the urine fell in quantity,

and in four days the pain was so distinctly localised in the right kidney, which had never previously been affected, with at the same time almost complete suppression of urine, that right lumbar nephrotomy became necessary. Before proceeding to this, however, the wound in the left loin was again laid open, and several small calculi were removed. The right kidney was found surrounded by pus, there was pus in the pelvis, and the ureter was blocked by calcareous concretions which the operator succeeded, by the use of a syringe, in washing down into the bladder. A like experience befell Mr. Lucas in having to perform nephrolithotomy on a patient's left side, four months after the removal of the right kidney for calculous pyelo-nephritis of long standing.

More striking than these cases is the case of Lucas-Championnière (recorded in Brodeur's work, p. 352). A woman, æt. 42, had been passing calculi and suffering severe left lumbar pain for eleven years. In November, 1884, a small calculus was removed from the bladder, but there was no marked cessation of her other symptoms. In November, 1885, there was more severe pain in the left loin, and only a few drops of urine were passed in the twenty-four hours. Then for four days there was complete suppression. On the 21st a few drops were passed, and then again for four days there was no urine. Such at any rate was the story when she was admitted into hospital on November 30th, with high temperature and frequent vomiting. There was no urine, and Lucas-Championnière diagnosed obstruction of the left ureter, and "reflex anuria" on the opposite side. An exploratory loin incision was made on December 5th, when the finger opened a large abscess in the kidney substance, but no calculus was found. There was much flow of urine from the wound, but not until twenty-one days after was there any passage by the urethra. On December 30th urine ceased to flow from the side. Early in January (1886) a calculus was detected in and removed from the bladder, and later in the same month she had a return of

severe lumbar pain with high temperature, with the passage of a long blood-clot but no stone. A case like this is of much importance in considering the question of sympathy between the kidneys, although I cannot refrain from expressing grave doubts as to this patient having had a right kidney at all. If "reflex anuria" be the true explanation of the suppression, which clearly was of the "obstructive" variety in Lucas-Championnière's case, it is to say the least remarkable that reflex suppression is so rare a thing in cases of calculous pyelo-nephritis. And this appears the more remarkable when we remember that the conditions predisposing to the formation of stone must affect both kidneys alike, and that when there is certainly a calculus on one side, there is some, though it may not be very strong, presumptive evidence that there is a calculus on the other, and that the other kidney, even though there be an absence of the symptoms of stone in it and it be doing its work well, may be in a condition, not perhaps of active disease, but in a condition in which disease may be very readily established. Was that the state of things in the case recorded to-night, and in others like it, or had the affection of the one kidney which was known to be diseased no influence whatever, neither direct nor indirect, on the later affection of the other kidney which had previously shown no symptoms at all? No certain answer can be given, but whatever be the true explanation the facts of the case remain, and have an important bearing on renal surgery, especially on the operation of nephrectomy, and on preliminary abdominal exploration for determining the state of each organ.

Had I performed nephrectomy in this case I know now that I should have done very wrong, but I have never quite been able to satisfy myself that had the abdomen been explored, and we had found the left kidney much atrophied and with a huge dilated pelvis, seemingly in such a condition as to suggest, as indeed at a future time it was suggested, that it was a worthless organ and had

better be removed, and we had at the same time felt that the right kidney was healthy, if such a thing can be determined by the feel, that it was moreover enlarged sufficiently to do the work of two, and there had been no discovery of the small calculi which it contained, we might not have been led to take the left kidney away. And thereafter might have arisen that event which has been recorded in the history, when the right kidney was in its turn placed *hors de combat*, was inadequate in fact for the needs of the individual, and the left, the atrophied and seemingly useless kidney, had to carry on the work of both glands. Is it as likely that the patient would have been alive now if the left kidney had been removed at the time of the first operation?

Abdominal exploration may be a good thing, but it may be a very evil thing if there be a neglect of the considerations which are suggested, not I think without some warrant, by the case brought before the Society to-night, for the surgeon may be led by it to the removal of an organ which the patient at some future time may sorely need.

In some very interesting "Reflections suggested by a Series of Cases of Renal Calculus," Mr. Godlee ('Practitioner,' Nov. and Dec., 1887) points out that we must not be in too great a hurry in urging a patient to submit to the removal of a kidney; for it must be remembered that though the organ may be a source of discomfort and of possible danger, it still does a certain amount of work. "It is exceedingly difficult," he says, "to estimate the exact condition of that of the opposite side, and I have shown elsewhere (making reference here, he is, to the case brought before this Society last session) that disturbance occurring in or in the neighbourhood of a much-diseased kidney may cause great functional derangement, amounting even to complete stoppage of that of the other side, although the latter may be much less diseased, in fact, comparatively speaking, healthy."

This question of the supposed sympathy between the

kidneys is clearly a most important one, and needs much further elucidation. If I needed an excuse for bringing this case before the Society I should say that we do not by any means know all that there is to be known; and with Mr. Godlee, I will add that "we have yet to learn a good deal about the history of patients who are left with only one kidney" ('Practitioner,' Nov., 1887, p. 329).

*Note.*—The patient was present when this paper was read, and was then in every respect in perfect health.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 367.)





# REMARKS ON SPLENECTOMY, WITH A REPORT OF A SUCCESSFUL CASE.

BY

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THE subject of excision of the spleen does not appear to have been brought before this Society until April, 1886, when two cases of splenectomy formed the subject of a paper by Mr. Knowsley Thornton which is published in vol. lxix of our 'Transactions.' At the close of that paper Mr. Thornton gives tables of all the cases he could find recorded up to that time. And we learn that there had then been eleven cases of complete splenectomy which were successful, four other successful cases of partial removal of injured spleen, and twenty-four unsuccessful splenectomies. Among the latter were three of my own cases—one, the first in Great Britain, in 1865, the second in 1868, the third in 1876. An account of all these cases may be found in my last work on abdominal tumours, published in 1885. I did not remove an enlarged spleen after the case in 1876 until last year, when I performed the operation which I have now the honour of submitting for the consideration of the Society.

In October, 1885, by desire of Dr. Morley Rooke, of Cheltenham, I first saw an unmarried lady, then twenty-two years of age, on account of an abdominal tumour about the size of a young child's head. The tumour was below the umbilicus, central, elastic, but without fluctuation. Its movements were closely associated with those of the uterus, and its lower border was felt behind and to the left of the uterus. I was doubtful as to the precise seat and character of the tumour, but thought it must be either uterine or ovarian, a very florid complexion rather inclining me towards the former view.

I was informed that since the age of nine years the young lady had been subject to frequent attacks of jaundice, and the mother said that the urine had "always" been very dark in colour. The catamenia appeared rather late—after fourteen—and were at first very irregular. Until after fourteen she was very thin, but then began to grow stout, and her mother soon became uneasy at her being "so large in front." In January, 1882, she had scarlatina very severely, and was long afterwards in delicate health, but did not complain of abdominal pain until after an attack of diarrhoea in August, 1885. It was then that Dr. Rooke first examined the abdomen, and he writes to me that he then "found a distinct pelvic tumour of some sort—central and occupying exactly the position of a uterine fibroid. On vaginal examination, although the tumour could not be felt, yet, with the other hand pressing over the abdomen, distinct impulse was communicated to the cervix." He adds, "I did not use the uterine sound. The growth, when manipulated through the abdominal walls, was not readily movable laterally—presumed size that of a seventh month foetal head. My diagnosis was either an ovarian or a uterine tumour, most probably the latter." As I have just said, I entirely concurred in this view of Dr. Rooke's, and he carefully watched the case without any treatment beyond alkaline diluents. The tumour slowly increased in size, and Dr. Rooke says, "I then resolved to try

ergot, and she took this internally for a considerable period. Under it the growth at one time certainly decreased."

I saw her occasionally at intervals of a few months, and learned that she pursued rather an active life, driving out daily, joining parties of various kinds and dancing, an occasional attack of jaundice being the chief subject of remark. In November, 1886, I found the tumour very much as when I first saw her, and the ergot was discontinued, having been taken eight months. She then went to Italy for the winter, travelled a good deal, but was very easily tired. In January, 1887, in Rome, she over-fatigued herself, working three hours a day, standing in an artist's studio. Then she went through a good deal of fatigue, travelling in Sicily, and afterwards round Naples. On the homeward journey she was treated by Dr. St. Clair Thompson, at Florence, for an attack of peritonitis with very severe abdominal pain, but was able, after a fortnight, to continue her journey. In July, 1887, finding the tumour somewhat increased in size, I agreed with Dr. Rooke that she should take ergot again. This was continued for six weeks. By this time she had become anxious to have the tumour removed, but we thought the suffering hardly sufficient to justify so serious a risk, as she was able to enjoy driving and ordinary amusements. In August and September, 1887, she had a severe attack of jaundice with much abdominal pain. In October, when at Cambridge, Dr. Latham attended her in another attack. I was told that her skin was then "quite copper-coloured," and Dr. Latham wrote to me that the illness "was marked by sudden and intense discolouration of the skin and conjunctivæ, extremely dark urine, obstinate constipation, and the same severe abdominal pain which had accompanied the previous attack." From about this time a considerable change was observed in the tumour. It rose from the pelvis, increased in size, was more mobile, and fluctuation became more perceptible in or around it; and she was suffering

so much, not only from the pressure of the tumour, but in her general health, that I quite agreed with Dr. Latham in advising that the tumour should be removed. I wrote to Dr. Rooke to this effect, advising that if the suffering continued, operation should not be long delayed; and Dr. Rooke, finding the changes that Dr. Latham and I had observed, fully concurred in our opinion and advice.

Accordingly the patient came to London, and I operated on December 5th, 1887.

All the usual antiseptic precautions were adopted, including phenol spray. Dr. Day administered bichloride of methylene, and I was assisted by Mr. Meredith and Dr. Rooke, Dr. Headley, of Melbourne, being the only visitor. The incision was made in the median line, between the umbilicus and symphysis pubis. As soon as the peritoneum was opened, about two or three pints of ascitic fluid escaped, and the spleen was seen at once. Passing one hand around it, I felt that the uterus and both ovaries were of normal size, and that the spleen was movable. I enlarged the incision until it extended from one inch above to about five inches below, the umbilicus. On attempting to press out the spleen with one hand passed behind it, an accidental rupture was followed by very free bleeding. The substance of the enlarged organ was very soft and friable, so I brought it out as soon as possible, and put two pressure forceps of large size on the connection between the spleen and the gastro-splenic omentum, which was unusually fatty and long, and contained very large blood-vessels, also much elongated. Transfixing twice behind the forceps, each needle with a double ligature, the connection or pedicle was tied in four parts. The pressure forceps were then removed. One ligature was applied behind the four, securing the whole of the pedicle, and a second ligature was applied as additional security upon the largest vessel. The peritoneal cavity was then carefully cleansed by sponging. A good deal of blood was lost; but it was venous or very dark, and appeared rather to come from the substance of

the spleen than from the general circulation. The cheeks and lips never lost their florid colour, nor did the pulse become weak.

The wound was closed by silk sutures, as in ovariectomy, and the abdomen supported by plaster, salicylic wool, and a flannel belt.

Mr. Eve examined the tumour at the College of Surgeons the same afternoon, and reported it weighed 1 lb. 14 oz. This was after much blood (variously estimated at from 3 lbs. to 5 lbs., but not measured) had drained out of it. The blood contained an excess of white corpuscles, but not to a remarkable amount. Mr. Eve had not the necessary instrument for making an exact estimate.

There is not much to remark on the progress after operation; except that until the catamenia commenced on the fourth day there was high temperature, flushed face and forehead, very scanty urine, and dry skin. I should have stated that the operation was performed about a week after the non-appearance of an expected menstrual period. I proposed delay, but was assured that whenever she had missed a period before it had not appeared until the next was due, and three weeks' delay was more than she could safely bear. Before the operation the temperature was normal. It rose within eight hours to  $101^{\circ}$ , the next day to  $102.6^{\circ}$ , the second day to  $103.6^{\circ}$ , in spite of an ice-cap to the head, and remained between  $101.0$  and  $102.4^{\circ}$ , with a pulse of 120 to 128, until the evening of the fourth day, when menstruation came on. This was soon followed by falling temperature and slower pulse. After the fifth day the temperature was almost normal and the pulse about 80. The urine became more copious and the skin moist. Neither sickness nor pain had ever been troublesome. The stitches were removed on the seventh day, when the wound was perfectly united by first intention, without a drop of moisture from any of the stitch points. She left London twenty-four days after operation and bore the journey to Cheltenham perfectly

well. Writing to me on the 13th of January, 1888, Dr. Rooke said, "Since her return home she has continued to improve, is driven in an open carriage, walks about the house, eats and sleeps well, retains her vivid colour, and so far seems to do better without a spleen than with one. There is no appearance of jaundice. I question whether she has, for many years past, been six weeks without an icteric tinge."

A little consideration of Mr. Thornton's tables is sufficient to prove that the results of splenectomy vary very much with the conditions which have led to the operation,—that it is very successful in cases of partial removal for injury, less so in cases of wandering spleen and of splenic cysts, still less so in cases of hypertrophy, and is very unsuccessful when the hypertrophy is associated with leucæmia.

In the fifth volume of the 'Transactions of the American Surgical Association,' published at Philadelphia in 1887, there is a paper by Dr. McCann, of Pittsburg, on "Splenectomy for Dislocated or Wandering Spleen." He records a successful case in a married woman, twenty-nine years of age. The spleen was in the left iliac region and "extended into the pelvic cavity. It was removed in May, 1886, and weighed fourteen ounces, after a quantity of blood had been allowed to escape from it." . . . . "The patient's blood contained more than the normal number of white blood-corpuscles, but the number was not sufficient to constitute leucæmia." The patient recovered, became pregnant five or six months after operation, and when the paper was read or published, she was in the eighth month of pregnancy and had "been able to perform without discomfort all the duties incumbent upon a farmer's wife."

From a comparison of different compilations of published cases, Dr. McCann concludes that complete excision of the spleen for all causes has "been performed fifty-two times, with a mortality of 62·8 per cent. But while in sixteen cases where hypertrophy of the spleen was

associated with leucæmia every patient died, thirteen operations gave eight recoveries where the spleen was excised for injury, for hypertrophy uncomplicated with leucæmia, or for dislocated or wandering spleen."

In the thirty-sixth volume of Langenbeck's 'Archiv' there is a very learned and valuable paper by Professor Adelman, on the progress of splenectomy in thirty years. He has arranged in a table twenty-five cases of recovery where the healthy spleen has been removed, it having escaped from the abdominal cavity after wounds or accidents of various kinds. He does not attempt to tabulate fatal cases, as he very justly says such cases are seldom recorded. Some of these twenty-five successful cases prove that life may be prolonged and good health enjoyed for many years after the loss of the spleen. In another table he gives particulars of fifty-four cases of splenectomy where the spleen was enlarged or diseased, bringing the record down to June, 1887. He corrects many errors in the tables previously published, and with some doubtful exceptions concludes that the operations recorded before 1848 were performed without any anæsthetic, and most of those before 1875 without anti-septic precautions. He shows that of the fifty-three patients, only eleven were males to forty-two females. The ages varied from ten to sixty-three years, but age alone did not appear to have any great influence on the result. The nature of the disease is far more important, as shown by the following table :

Disease.	No. of cases.	Recoveries.	Deaths.
Hypertrophy with leucæmia . . .	19	1	18
Simple hypertrophy . . .	14	1	13
Malarial hypertrophy . . .	4	1	3
Wandering spleen . . .	9	7	2
Splenic cysts . . .	4	3	1
„ hydatid . . .	1	1	0
„ sarcoma . . .	1	1	0
„ suppuration . . .	1	1	0
	<hr/> 53	<hr/> 16	<hr/> 37



It might appear from this table that the result of splenectomy for simple or malarial hypertrophy, and especially where the enlargement is associated with leucæmia, is so disastrous that the operation should be generally discouraged. But when we examine into the causes of the great mortality, it is almost certain that more extended experience, and more attention to the manner of securing blood-vessels, may be expected to lead to much greater success.

One question of great practical importance was raised by Langenbeck. He suggested that the splenic artery should be tied as close to the celiac axis as possible, before it divides into its numerous branches. But even if this could often be done without much disturbance of the pancreas, it must be very doubtful whether a ligature applied close to the celiac axis in a patient whose blood is in a condition where a firm blood-clot can hardly be expected, can be trusted not to slip and lead to fatal secondary hæmorrhage. From my own experience and study of many of the records of operations by other surgeons, I am inclined to follow and recommend the opposite practice, temporarily securing all the blood-vessels by pressure forceps as near to the spleen as possible, then removing the enlarged organ, afterwards applying silk ligatures by transfixion behind the forceps, and tightening them as the forceps are removed. All the ends of the silk should be cut off near the knots. If the peritoneal cavity is carefully cleansed, there can be no necessity for drainage, and the wound in the abdominal wall should be closed as in ovariectomy.

It may still be open to question whether splenectomy should be advised in cases of simple or hydatid splenic cysts. So far as one perfectly successful case which I treated in association with Sir William Jenner, by tapping and drainage, can guide us, I should be disposed to advise a trial of the simpler process before resorting to the graver operation.

When the spleen is hypertrophied, the danger of

removal is certainly less when the organ is also misplaced or "wandering" and its attachments lengthened. And it is probable that a spleen simply hypertrophied without leucæmia may be removed with less risk than when leucæmia is present. It is also probable that the amount of the leucæmia, or the larger or smaller proportion of white to red corpuscles in the blood, may have a considerable influence upon the amount of blood lost during the operation and upon the liability to secondary hæmorrhage, and thus affect the result.

It must also be admitted that under very different conditions of the patients the results of splenectomy have gone on improving during the last thirty years, and that with increasing knowledge and more practical experience, still greater success may be reasonably hoped for. And sufficient facts have already been accumulated to prove that patients who recover from the operation may live for many years in good health without a spleen.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 368.)



A FURTHER CONTRIBUTION  
TO THE STUDY OF  
RHEUMATOID ARTHRITIS.

BY  
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IN a former paper I endeavour to show, from the study of the statistics of 500 cases of rheumatoid arthritis, the truth of the statements made by certain authors, that the causes of rheumatoid arthritis are such as might be expected to act upon the central nervous system, and that the distribution of the lesions, and the order of invasion of the joints, lend support to the theory of the nervous origin of this disease.

Continuing the subject I propose, in the present paper, to examine certain other phenomena of rheumatoid arthritis, with a view to seeing whether or not their occurrence supplies any further evidence of the truth of that theory.

In many cases muscular atrophy, the condition known as "glossy skin," as well as certain sensory disturbances, are met with, and the question is whether these conditions

are to be regarded as integral parts of the disease, or whether they are merely secondary changes, following upon the damage to the joints, not resulting from disease, but exemplifying the general law that the irritation of articular disease of any kind is communicated to the spinal cord, and so brings about reflex changes, such as reflex muscular atrophy, and a spasmodic condition of the muscles in the neighbourhood of the affected joints.

Dr. Ord is inclined to take the former view in regard to some cases of the disease, and in his address to the British Medical Association at Belfast, speaking of a *certain group* of cases of rheumatoid arthritis, he said:—“In fact as they present themselves to me, these are cases of progressive dystrophy of joints, marching with progressive atrophy of muscles, and with atrophy of other tissues of the limbs.”

Dr. Charcot, on the other hand, regards the muscular atrophy of rheumatoid arthritis as merely an example of arthritic muscular atrophy, more marked than usual, in this disease, on account of the severity and long duration of the joint lesions.

The demonstration by MM. Pitres and Vaillard<sup>1</sup> of the fact that peripheral neuritis is present in some of these cases, suggests that the changes in the nerves may be the cause of some of these dystrophies.

These observers find that the peripheral nerves are the seats of important changes in cases of rheumatoid arthritis, and they have met with such changes in all the three cases which they have examined, but that the neuritis does not apparently play a primary part in the production of the articular lesions, since the nerves supplying the affected points may exhibit only trifling changes. They regard it as the probable cause of some of the sensory symptoms, and as standing in a constant relation to the muscular and cutaneous atrophies.

This explanation of the production of the dystrophies is opposed by the fact that, in rheumatoid arthritis, the

<sup>1</sup> ‘Revue de Médecine,’ June 10th, 1887.

tendon reflexes are usually exaggerated, and this exaggeration is frequently well marked when the muscles are conspicuously atrophied, a fact hardly consistent with the production of the atrophy as a result of peripheral neuritis. On the other hand, increase of myotatic irritability is a characteristic of all forms of arthritic muscular atrophy, being well marked in cases in which injury to joint has been followed by the wasting of neighbouring muscles. Moreover, the increase is not confined to the affected limb, both knee-jerks being not infrequently exaggerated when there is synovitis of one knee.

The occurrence of such increase of myotatic irritability in cases of rheumatoid arthritis is therefore only what is to be expected if the atrophy of the muscles is merely secondary to the joint lesions; but, on the other hand, we have the fact that MM. Pitres and Vaillard found advanced changes in the nerves which supplied the atrophied muscles, and it is upon this fact that their view that the neuritis is the cause of the atrophy is based.

Although the increase of reflexes in this disease is widely recognised, I am not aware that any systematic examination of these phenomena in a number of cases has been hitherto carried out. I have therefore tested the jerks in fifty cases of rheumatoid arthritis which have recently passed through my hands, and the results are embodied in a table which is appended to this paper.

To ensure as much uniformity as possible, the supinator, triceps, and patellar reflexes were in each instance tested by the aid of a percussion hammer.

In some few instances the examination was precluded by stiffness and painfulness of the neighbouring joint, but usually no such obstacle was met with, and I do not think that, except where it is so stated, the state of the joints interfered in any way with the results obtained, since in cases where there was considerable enlargement present the reflexes were often found to be markedly increased. On glancing over this table it is at once apparent that there is little uniformity in the results obtained. In

apparently similar cases the jerks may be markedly increased in one, normal or diminished in another.

Nor are the results more uniform in individual instances. Patients who have all their limbs affected, apparently to a like degree, may have all the reflexes normal or diminished, with the exception of one or two, which are found to be greatly increased. In thirty-two out of the fifty cases there was definite increase of one or more of the reflexes.

Since it is difficult to appreciate the slighter degrees of muscular atrophy, especially when there is no opportunity of comparing the diseased with a sound limb, I have divided the cases into those in which the atrophy was sufficiently advanced to be noticeable on inspection and those in which it was not so.

Of twenty-three cases in which there was marked atrophy there were eighteen in which there was increase of some of the reflexes, whilst of twenty-seven in which the atrophy was not marked, there was increase of reflexes in fourteen only.

The amount of muscular atrophy varies very greatly, being hardly appreciable in some cases in which the joints are very seriously affected, whilst it is conspicuous in some of the early cases, in which the enlargement and deformity of the joints is comparatively slight.

When the cases are arranged in the order of their duration no definite results are arrived at, and this is not surprising when we call to mind the very different rates at which the disease advances in different patients. There are, however, a few observations which seem to show that when one limb is attacked before the corresponding one of the other side, the reflexes are greater on the side more recently affected.

One point, however, comes out clearly, namely, that as in other instances of arthritic muscular atrophy, the increased myotatic irritability is not confined to the affected limb. In cases in which one hip-joint only is diseased, both knee jerks-and even the supinator and triceps jerks as well may be increased. In some

instances the increase of the reflexes was very remarkable.

Thus in a man, æt. 32, who dated his disease from a prolonged immersion in the Orange River, in South Africa, two years previously, all the reflexes were greatly exaggerated. The joints affected were those of the hands and feet, the elbows, shoulders, ankles, and knees. A tap upon either of the supinator tendons caused contraction of the triceps, and a slight clonic spasm, and in the same way, when the triceps tendon was struck, clonus was observed, and all the arm and scapular muscles contracted. The patellar reflexes were also greatly increased and slight ankle-clonus was obtained on the left side. There was considerable deformity of the affected joints, and great wasting of muscles of all the limbs.

The notes of a still more remarkable case have been published by Dr. Mader,<sup>1</sup> of Vienna.

The patient was a woman, æt. 43, who had suffered from rheumatoid arthritis for fifteen years. The nerve-trunks of the upper extremities were tender on pressure. A tap on the patellar or triceps tendon not only elicited a greatly exaggerated reflex but also caused contraction of the trunk-muscles.

The left arm was always jerked when the right was tapped, and the same result could be produced by a slight blow upon the right tibia, or over the sternum. Such experiments always started a clonic spasm of the whole of the body, lasting several minutes.

How are we to explain the want of uniformity in the results obtained from the examination of the fifty cases?

Probably in some instances the deficiency of the jerks is due to the advanced changes which have taken place in the substance of the muscles.

Is it possible that the changes in the peripheral nerves may act as a disturbing factor in some instances, neutralizing the tendency to increase of the reflexes?

<sup>1</sup> 'Berichte der k. k. Kraukenanstalt, Rudolph Stiftung in Wien,' 1881, p. 266.



Perhaps we may look upon the tingling and numbness in the affected limbs, phenomena which are not infrequently met with in course of rheumatoid arthritis, as symptoms of the same nerve changes.

These symptoms have received but little attention from those who have written upon the subject. They are noted in some of the published cases, but I am only acquainted with the two following distinct references to their occurrence.

Dr Homolle, in his article in the '*Dictionnaire de Médecine et de Chirurgie Pratique*,' says:—"Anomalous sensations of numbness and formication, and vague pains in the limbs, are usually the first symptoms of which the patients complain," and he compares them to similar sensations met with in connection with certain spinal diseases.

Dr. Howard again, in '*Pepper's System of Medicine*,' writes as follows:—"For weeks or months the patient may experience numbness or tingling and rheumatic pains in the limbs, perhaps with a sense of stiffness in the joints, especially felt after rest, or the day after unusual fatigue."

I have come across several cases of this kind lately, and it certainly appears that, as these authors remark, these symptoms occasionally precede the joint lesions, and have the appearance of being primary symptoms of rheumatoid arthritis.

Thus a married woman, æt. 52, had had a sensation of pins and needles in the hands and arms for two years, accompanied by gnawing pains in the bones, yet the joints of the fingers had only been affected for one year.

Another female patient volunteered the information that the disease came on with tingling in the fingers, like pins and needles, and when these symptoms first appeared she had violent pains in both arms, lasting for three or four hours, and followed by sweating.

In other cases the tingling and numbness appear

simultaneously with the joint affection, and in others again they come on at a later period of the disease.

Tingling and numbness are prominent symptoms of peripheral neuritis, and it is curious to note that in no case in which a history of such symptoms was obtained did I find any increase of the tendon reflexes. The jerks were not, however, abolished, but were simply normal or diminished.

In considering any sensory disturbances met with in the course of rheumatoid arthritis, one source of fallacy must always be borne in mind. This disease not infrequently affects the joints of the vertebral column, causing that stiffness of the neck of which the patients so often complain. As Leyden and others have pointed out, this spondylitis may lead to pressure upon the nerve-roots, and so to radiating pains, and even to loss of power in the limbs. The following case, for the opportunity of seeing which I am indebted to Dr. Andrew, under whose care the patient was, appears to me to be one of this kind.

A young woman, æt., 24, was attacked in October 1885, with rheumatoid arthritis of the right wrist-joint. The disease soon involved the right foot, the knees, hands, shoulders, and temporo-maxillary joints. One night, in April, 1886, she was seized with severe burning pain in the right arm, extending from the shoulder to the fingers. This lasted about ten hours, and ceased as suddenly as it had commenced. The following morning she found that she had lost the use of the arm, and she had no feeling in it. Power and sensation returned in about a fortnight. During the same night her neck became stiff, and she began to suffer from pain along the vertebral column, and especially between the shoulders. At the time when I saw her there was well-marked grating felt when she rotated her neck.

Such causes will not, however, account for all the sensory symptoms met with in cases of rheumatoid arthritis.

The muscular atrophy of rheumatoid arthritis resembles

the other forms of arthritic muscular atrophy in other respects beside the increase of myotatic irritability. Its distribution is similar, the muscles which extend the affected joints suffering most. The hands of the patient have a peculiar appearance, owing to the wasting of the interossei, especially the abductor indicis, and this peculiarity is often increased by the enlargement of the knuckle-joints.

The electrical reactions resemble those of ordinary arthritic atrophy, the reaction of degeneration being rarely present, and when found being as a rule limited to one or two of the muscles which are most seriously affected.

Associated with the wasting there is also a spasmodic condition of the muscles, which manifests itself in the form of severe cramp-like pains to which these patients are peculiarly liable, and which cause the most acute suffering which they endure, and gives rise at a later period to those distortions which constitute the most serious deformity met with in rheumatoid arthritis.

Moreover, the muscular atrophy follows the appearance of the joint disease after a greater or lesser interval. In ordinary cases of rheumatoid arthritis we do not find the extension of the disease to a fresh joint heralded by the atrophy of the muscular structures which move that joint.

Dr. Ord is of opinion that these non-articular troubles do sometimes precede the appearance of the joint lesions, and it is certain that articular changes, clinically identical with rheumatoid arthritis, are sometimes to be met with coming on as a later affection, in cases in which other dystrophic phenomena have been present for some time previously. The question is, whether such cases can be regarded as examples of ordinary rheumatoid arthritis, or whether they are not rather arthropathies of nervous origin, in the more limited sense of the term, lending powerful support to the nervous theory of rheumatoid arthritis by bridging over the interval separating that disease from the arthritis associated with spinal injuries and definite spinal diseases.

The following case may perhaps be regarded as one of this character.

The patient was a man, æt. 55, who had formerly been a sailor, but who had left the sea, and was engaged in the manufacture of rigging. He complained of pain in the region of the right clavicle, and down the right arm, from which he had suffered for some three or four months.

The right shoulder appeared somewhat flattened, and the muscles of the upper arm were peculiarly flabby. When requested to extend the elbow he did not bring the long head of the triceps into action at all, but the muscle acted well when extension was resisted. No difference could be detected in the measurements of the circumference of the two arms.

The patient complained of tingling and numbness of the right arm, with some loss of power, and the grip of the right hand was weaker than that of the left. The supinator reflex was increased in the affected arm, but the triceps reflex was not obtained. Fibrillary twitchings were noticed in the muscles of both arms, but were most marked in the right.

Electrical examination showed that faradic contractility was diminished in the right biceps and triceps, but was increased in the muscles of the thenar eminence.

Galvanic irritability was also increased in the thumb-muscles, and greatly increased in the triceps, but K. C. C. was everywhere greater than A. C. C.

There was, moreover, a painless affection of the carpo-metacarpal joint of the right thumb, with excessive mobility of the joint, grating of the articular surface, and enlargement of the heads of the bones.

The question of the nature of the muscular atrophy of rheumatoid arthritis is one of great importance from a theoretical point of view, for if we admit that it is an integral part of the disease, advancing *pari passu* with the joint lesions, we are almost bound to acknowledge the nervous origin of the arthritis, whereas if it be merely a

secondary phenomenon, simply due to the irritant action of the process going on in the joints, it can throw no light on the etiology of that process, and can supply no argument either for or against the nervous origin of the disease. To me, at least, it seems that we have no reason to regard this muscular atrophy as anything but a secondary process, not peculiar to rheumatoid arthritis, and therefore as giving no support to the theory under discussion.

The deformities met with in this disease may be grouped under three heads.

Firstly, there are those produced by osteophytic outgrowths around the joint, which are almost peculiar to this disease. I say almost, because similar enlargements are sometimes met with in gouty patients.

Secondly, there is the deflexion of the fingers to the ulnar side of the hand, which is in no way peculiar to rheumatoid arthritis, being a common phenomenon in gout, whilst the most extreme ulnar deflexion that I have ever seen was in a patient who had suffered from several attacks of rheumatic fever but whose joint showed no signs of any permanent change. This deformity I cannot regard as due to muscular action, for it is absent where the true muscular deformities are present, without joint lesions, and I would attribute it rather to the relaxation of the fibrous structures of the metacarpo-phalangeal joints, allowing the digits to fall outwards.

Lastly, there are the true muscular deformities of the hands and feet, which have been so minutely and accurately described and classified by Dr. Charcot. They also are in no way peculiar to rheumatoid arthritis, but may be met with in the course of nervous disorders, where there is no joint affection at all; and may also result from repeated attacks of gout in the hand or foot.

These distortions fall under two main types, which have been called by Dr. Charcot the types of extension or flexion.

The essential character of the type of extension is the hyper-extension of the second row of phalanges upon the

first, which is accompanied by more or less flexion of the other hand-joints ; whereas in the type of flexion the second row of phalanges is flexed upon the first, with hyper-extension of the other joints.

Each main type is capable of modification forming sub-types, and both may be present in different fingers of the same hand. Which type is produced in any particular instance is probably determined by the condition of the interossei, which little muscles are greatly affected by the atrophic process. When they are strong the type of extension results, but when they are overcome by the spasm of the other muscles the hand assumes the type of flexion.

The deformities are never met with in rheumatoid arthritis when the joints of the hands have escaped the disease, and, like the muscular atrophy, they are secondary changes, and no more integral parts of the disease than talipes is a part of infantile paralysis.

Well-marked changes in the skin and nails, with atrophy of the subcutaneous tissues, are much rarer than the muscular atrophy, but when they occur may be developed early in the disease soon after the neighbouring joints have been attacked.

In each of the three cases examined by MM. Pitres and Vaillard such changes were present, associated with peripheral neuritis ; the skin being most affected in the case in which the cutaneous nerves had suffered most. It seems probable therefore that these dystrophies may result from the peripheral neuritis, but whether the neuritis is a primary or secondary phenomenon of rheumatoid arthritis is a question which cannot be answered without further investigation. In other articular diseases peripheral neuritis is met with as a secondary phenomenon, being probably caused by the extension of the morbid processes from the joints to the nerves, but the comparative immunity of the articular nerves in the cases examined is opposed to this explanation.

In the following case the skin change was apparently

due to a mere extension of the inflammatory process to the nerves, since it was confined to the peripheral side of the seats of disease.

A young married woman, æt. 22, who had no family history of articular disease, and who had never suffered previously from any joint affection, was attacked during her second pregnancy with rheumatoid arthritis of the small joints of the carpus of each hand, and, to a much less extent, of the tarsi. When seen the skin of the hands was glossy and atrophied but not pink. This change did not extend at all beyond the seats of the disease. The nails had not undergone any dystrophy. She had felt numbness and tingling in the same areas soon after the joints were attacked, and had noticed the glossiness of the skin since the same period. I could not detect any loss of sensation in the fingers. On the right side the supinator jerk was poor; on the left it was good, but not exaggerated. Both triceps-jerks were well obtained, and the knee-jerks were normal.

The conclusions are as follows :

1. Although lesions identical with those of rheumatoid arthritis are occasionally met with in cases in which muscular atrophy and other indications of nervous disorder have previously existed, the ordinary muscular atrophy of rheumatoid arthritis is merely an example of reflex arthritic muscular atrophy, with which it agrees in its distribution, and in its association with increased myotatic irritability and muscular spasm.

2. That the remarkable distortions met with in this disease, being due to this muscular spasm, are merely secondary phenomena; and being, like the muscular atrophy, in no way peculiar to rheumatoid arthritis, can supply no argument for or against its nervous origin.

3. That peripheral neuritis, which has been shown to be present, plays an as yet undetermined part in the production of the phenomena of the disease; that it may perhaps be responsible for the irregular results of examination of the tendon reflexes; that it may give rise to

certain sensory phenomenon which are often early, if not primary symptoms; and probably is the cause of the dystrophic condition of the skin and nails occasionally met with.

### TENDON REFLEXES IN RHEUMATOID ARTHRITIS.

*Cases in which all the Reflexes were Exaggerated.*

No.	Sex and age.	Duration of disease.	Joints affected.	Condition of reflexes.	State of muscles, &c.
1	M. 31	4 months	Hands, wrists, ankles, neck, jaw	Supinator much increased; triceps much increased; knee increased	No well-marked atrophy.
2	F. 55	2 years	Hands, wrists, elbows, right shoulder, left hip, knees, sterno-clavicular	Supinator, left increased, right much increased; triceps, left increased, right much increased; knee much increased	Well-marked atrophy, especially in right arm.
3	F. 35	1 year	Hands, wrists, knees	All reflexes increased	Well-marked atrophy, especially in arms.
4	F. 66	12 years	Hands, feet, knees, elbows, hips, ankles, back	All reflexes much increased	No well-marked atrophy.
5	F. 45	1 year	Wrists, hands, feet, ankles, knees	Supinator much increased; triceps increased; knee much increased	Atrophy not well marked; distortion of hands, type of flexion.
6	M. 32	2 years	Hands, elbows, shoulders, feet, left ankle, knees	Enormous exaggeration of all the reflexes; clonic spasm	Much atrophy.
7	M. 45	6 months	Right hip	All reflexes increased	Wasting of muscles around right hip-joint.
8	F. 30	R. wrist 14 years; L. wrist 1 year	Both wrists, right stiff	Supinator, left increased; triceps and knee increased, left triceps more than right	No marked atrophy.
9	F. 34	10 months	Hands, ankles, knees, shoulders	All reflexes increased	Well-marked atrophy.
10	F. 35	5 years	Knuckles, feet, ankles	All reflexes increased; knees slightly	Well-marked atrophy.



*Cases in which some Reflexes were Exaggerated.*

No.	Sex and age.	Duration of disease.	Joints affected.	Condition of reflexes.	State of muscles, &c.
11	F. 35	7 years	Left hand, right wrist, knees slightly	Supinator and triceps increased; knees normal, or perhaps increased	No marked atrophy.
12	F. 45	5 years	Hands, elbows, shoulders, feet, knees, neck	Supinator increased; triceps and knees poor	Much muscular atrophy; distortion of hands.
13	F. 49	2 months	Knees, left shoulder	Supinator good; triceps, right increased; knee much increased	No marked atrophy.
14	M. 34	2 months	Hands, wrists, elbows, shoulders, hips	All reflexes increased except right triceps	Well-marked atrophy.
15	F. 32	2 years	Hands, wrists, elbows, shoulders, feet, ankles, knees, neck, jaw	Right supinator reflex only, much increased	Fairly marked atrophy.
16	M. 34	3 or 4 years	Left hip, slight in right hip	Triceps and knee reflexes increased; left knee reflex greater than right	Much atrophy of left thigh.
17	M. 53	14 years	Hands, wrists, right elbow, left ankle, knees	Triceps reflexes much increased, especially right; knees could not be tested	Well-marked atrophy.
18	M. 43	6 or 7 months	Right hip	Both knee reflexes increased	Well-marked atrophy of right thigh.
19	F. 52	8 years	Hands, first right, then left; elbows; knees, right, then left; feet	Left supinator and triceps reflexes increased; knee reflexes both increased	Wasting, especially in right arm.
20	F. 35	8 years	Hands, feet, first right, then left	Right knee reflex increased; other reflexes normal or absent	Well-marked atrophy; distortion of hands and feet, type of flexion.

No.	Sex and age.	Duration of disease.	Joints affected.	Condition of reflexes.	State of muscles, &c.
21	F. about 40	1 year	End joints of fingers, right worst; right ankle	Right supinator and triceps reflexes increased; both knee reflexes increased	Slight atrophy.
22	F. 9	18 months	Hands, elbows, ankles, knees, neck	Knee reflexes increased	No marked atrophy.
23	M. 21	1 year	Right knee-joint	Left knee reflex increased; right could not be tested	Well-marked atrophy of right thigh.
24	F. 42	7 or 8 months	Carpometacarpal joints of thumbs, especially left	Left supinator reflex increased	No marked atrophy.
25	M. 60	1 year	Hands, right shoulder, right knee, slightly	Left supinator, both triceps, and left knee reflexes increased	No marked atrophy.
26	F. 37	4 months	Hands, shoulders, ankles, neck	Left supinator and both knee reflexes increased	Well-marked atrophy.
27	M. 45	4 months	Left hand and wrist, feet, ankles, knees, right hip	Left supinator reflex increased	No marked atrophy.
28	F. 46	1 year	Hands, knees	Right supinator, both triceps, and knee reflexes increased	No marked atrophy.
29	F. 21	7 years	Hands, wrists, elbows, shoulders, knees, hips, neck, jaw	Triceps and knee reflexes increased	Much muscular atrophy.
30	M. 36	7 years	Hands, feet, ankles, wrists, elbows, knees, hips, jaw	Right triceps reflex increased	Well-marked atrophy; distortion of hands, type of extension.
31	M. 66	7 years	Elbows, hands, wrists, left ankle	Knee reflexes much increased; supinator and triceps not obtained	No marked atrophy.
32	F. 54	2 years	Hands, wrists, knees	All reflexes increased except left knee	No marked wasting.

*Cases in which all Reflexes were Normal or Diminished.*

No.	Sex and age.	Duration of disease.	Joints affected.	Condition of reflexes.	State of muscles, &c.
33	F. 50	7 years	Fingers, knees, feet, ankles	Supinator and triceps reflexes absent; knee reflexes poor	Wasting not well marked (numbness and tingling).
34	F. 67	10 years	Hands, feet	Supinator and triceps reflexes absent; knee reflexes fairly good	Well-marked atrophy (numbness and tingling).
35	F. 25	20 months	Hands, feet, ankles, knees, elbows, neck, jaw	Supinator and triceps reflexes normal; knees could not be tested	Great muscular wasting.
36	M. 13	5 weeks	Hands, ankles, wrists, shoulders, right knee	Triceps reflexes absent; knee reflexes poor	Wasting not marked (numbness).
37	M. 48	5 or 6 months	Carpometacarpal joints of thumbs	Supinator and knee reflexes very feeble; triceps reflexes absent	No marked wasting.
38	M. 70	30 years	End joints of fingers	All reflexes absent	No marked wasting.
39	F. 31	18 weeks	Hands, knees, shoulders, elbows	All reflexes normal	No marked wasting (numbness and tingling).
40	F. 62	3 years	Hands, knees	Supinator reflexes poor; triceps reflexes absent; knee reflexes very poor	Some atrophy.
41	F. 55	2 years	Heberden's nodes	All reflexes normal	No marked atrophy.
42	M. 34	4 or 5 years	Left hip	All reflexes normal	No marked atrophy.
43	F. 62	17 years	Hands	Supinator reflexes slightly more than normal; triceps reflexes poor; knee reflexes normal, good	Well-marked atrophy (some distortion)
44	F. 67	20 years	Hands	All reflexes diminished	No marked atrophy.

No.	Sex and age.	Duration of disease.	Joints affected.	Condition of reflexes.	State of muscles, &c.
45	F. 52	2 years	Hands	All reflexes normal	No marked atrophy (tingling and numbness).
46	F. 25	14 months	Hands, elbows, feet, knees	All reflexes normal	No marked atrophy.
47	F. 37	5 years	Hands, jaw	All reflexes diminished	No marked atrophy (tingling and numbness).
48	F. 57	5 or 6 years	Right wrist, left elbow, and shoulder	Reflexes of upper extremities feeble; knee reflexes normal	Well-marked atrophy.
49	M. 34	About 3 years	Hands, wrists, shoulders, knees, hips, neck, jaw	Supinator reflexes normal; triceps reflexes obtained; knee reflexes poor	Much atrophy (distortion, type of extension).
50	F. 30	2 months	Hands, ankles	All reflexes normal	No marked atrophy.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 373.)



EFFECTS IN HEALTH AND DISEASE  
OF SOME  
DRUGS WHICH CAUSE RETENTION OF URIC ACID,  
IN CONTRAST WITH THE ACTION OF SALICYLATES, AS  
SHOWN IN A PREVIOUS PAPER.

BY  
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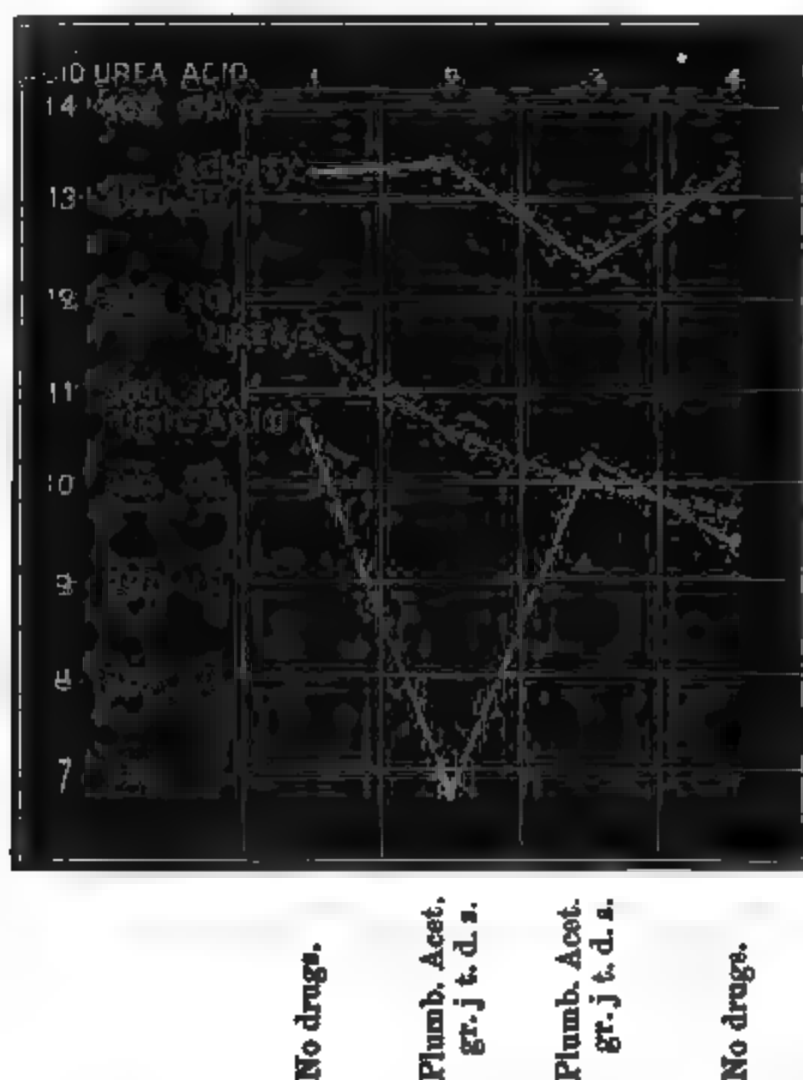
IN a paper recently read before the Society (January 10th, 1888) I have shown that a large part of the value of salicylic acid and its salts in disease is probably due to their facilitating the excretion of uric acid by forming a compound with it which is soluble in slightly acid fluids, that they thus not only directly increase excretion but also prevent acids from causing retention. For, as I have reason to believe that several of the more important diseases connected with uric acid are directly due to the retention of this substance in the body, occasioned for the most part by acids,<sup>1</sup> anything that will prevent acids from diminishing the solubility of uric acid and thus causing retention will have great power both to prevent and cure such diseases ; hence the importance which I attach to my results obtained with salicylates as brought forward in the above paper.

<sup>1</sup> ' Journal of Physiology,' vol. viii, Nos. 3 and 4.

I now propose to contrast with the above-mentioned action of salicylates that of several drugs which cause retention of uric acid, and to inquire how far such drugs are known to do harm in the diseases where salicylates are beneficial.

*Lead.*—It will be seen by the curves in Fig. 1 that the metal taken as the acetate causes clearly and distinctly

FIG. 1.



retention of uric acid, the uric acid falling below the urea to the extent of some three and a half grains on the first day of taking the drug ; and I may say that this experiment has been several times repeated and always gives this result. On the second day, however, uric acid rises above urea, and there is no retention, and this is the result

of several causes and does not in the least show any failure of action on the part of the lead. First of all, urea has fallen and acidity has fallen very considerably, and this alone would account for some rise of uric acid excretion. The fall of urea and acidity are probably due to the interference of the lead with intestinal digestive processes, and this also is a constant result of taking this salt. Again, as I have said in the 'Journal of Physiology,' acids appear to be able to cause retention only up to a certain point, and after some five grains or so have been deposited in the liver, spleen, &c., it is difficult to cause any further retention by the use of acids. Now, there is reason to think, from the curves shown, that at the time lead was taken in this figure there was already a considerable accumulation of uric acid in the liver, spleen, &c., so that both the causes I have mentioned may have prevented retention by lead on the second day it was taken.

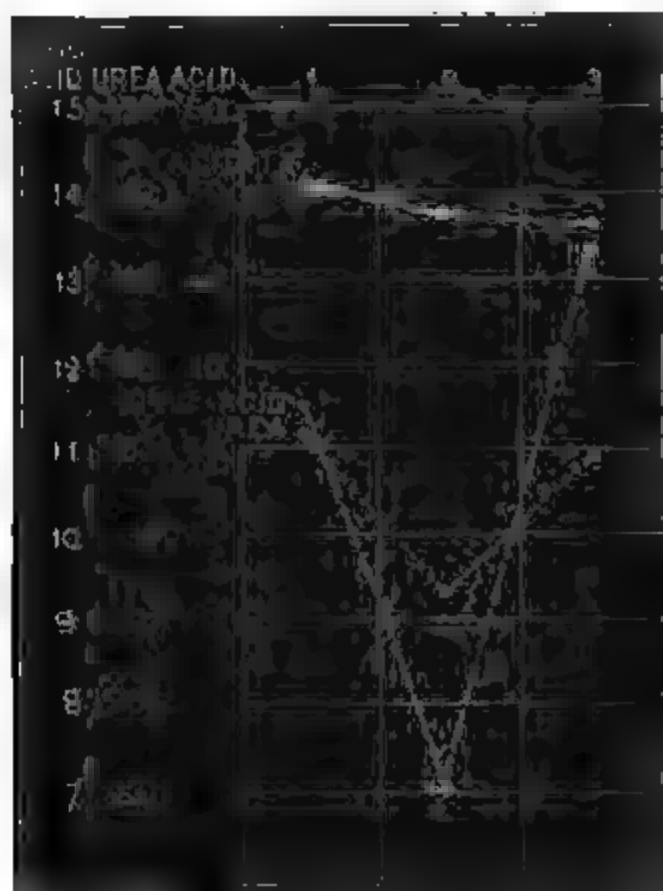
It may be asked why I did not continue the lead in the above curves so as to obtain further proof of its causing retention? My reason was this, that as soon as lead begins to interfere with intestinal processes so many factors are brought in that the problem becomes too complex to be of any value; thus the results of intestinal irritation, dyspepsia, flatus, diarrhoea, caused by any drugs of irritant nature, are a fall in urea and acidity. The result of falling acidity is a rise in the uric acid excretion, so that it becomes impossible to say what action the drug may have on uric acid excretion. There are several drugs which quickly cause intestinal irritation, and whose action on uric acid excretion (if any) it is therefore impossible to make out with certainty. This would be the case with lead except for the fact that it appears to act on uric acid excretion at once, but in the doses taken it only appreciably affects digestive processes after some twenty-four hours.

*Iron.*—It will be seen in Fig. 2 that this causes marked retention, but, unlike lead, its greatest effect takes twenty-four hours to come out. Possibly, the greater



solubility of the lead salt used may account for its quicker action. As soon as the iron is left off the uric acid rushes out in excess.

FIG. 2.



PIL. FERRI CARB.  
gr. i ter.

PIL. FERRI CARB.  
same dose.

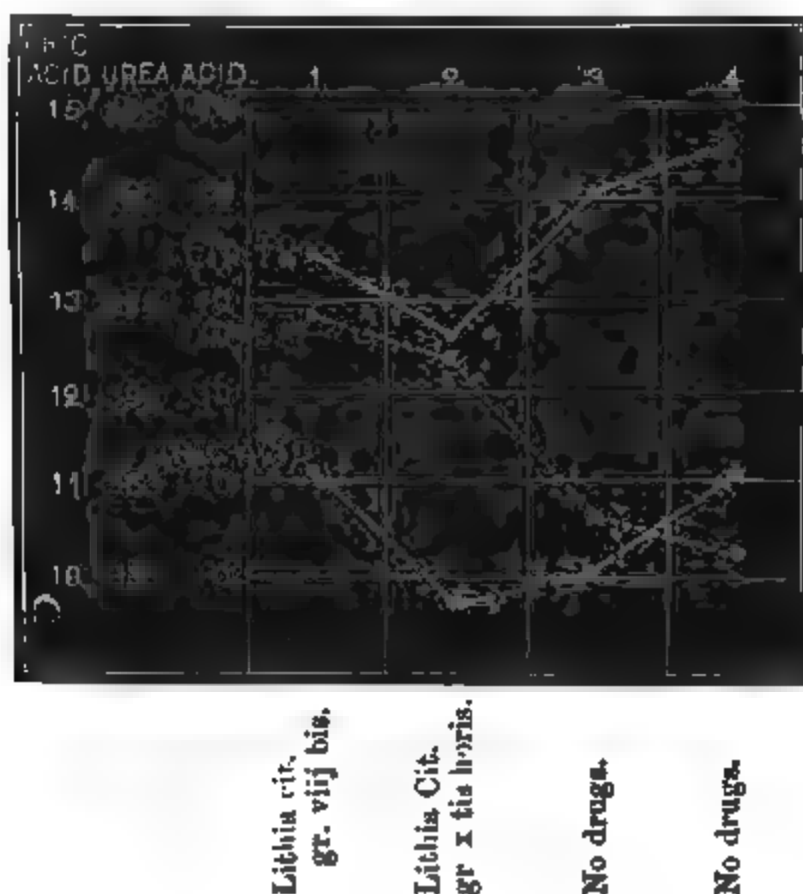
No drugs.

Urates of lead and iron are said by Sir A. Garrod to be quite insoluble; and this fact affords, I believe, the explanation of their action in causing retention. For, if salicylates form a soluble compound with uric acid and so facilitate its excretion, it is reasonable to suppose that if these metals form insoluble compounds with it they would prevent excretion and cause retention.

*Lithium.*—Here we have a drug whose urate has been shown by Sir A. Garrod to be extremely soluble, and yet

Fig. 3 shows, as I believe, that it causes retention of uric acid. When I first noticed this action of lithia salts I thought it must be an error, but repeated experiments showed me that the result was invariable ; and I was then

FIG. 3.



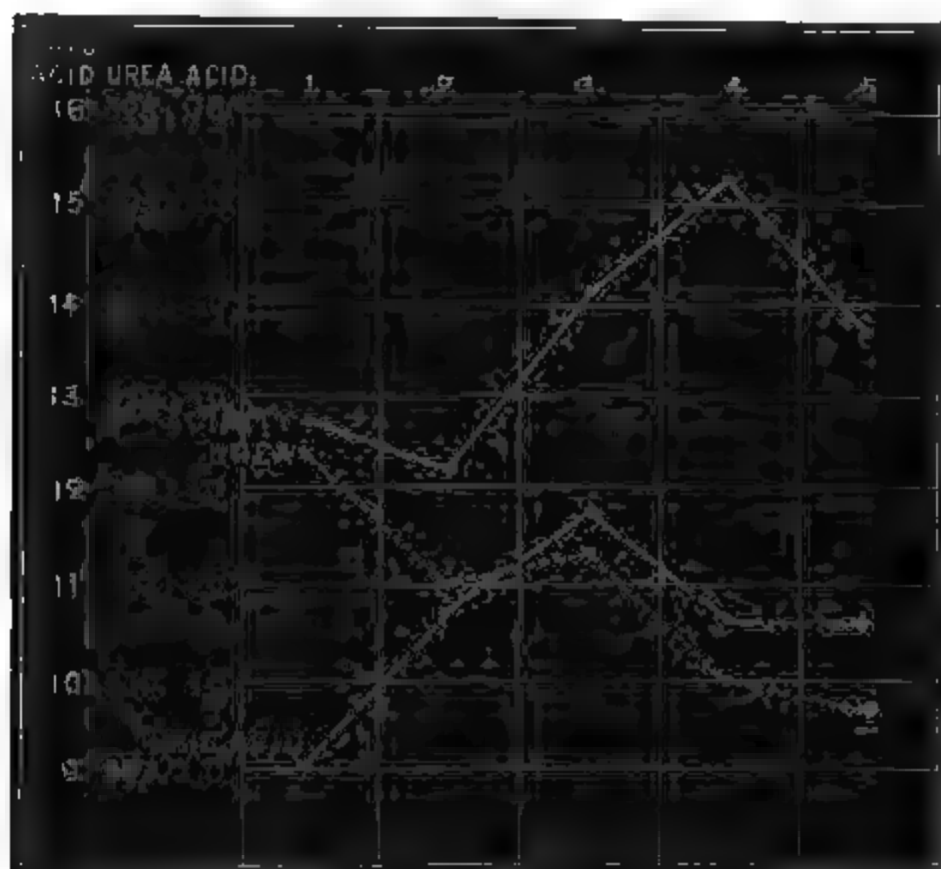
inclined to conclude that this was a fatal blow to the theory of the solubility of the compounds formed as the cause of uric acid excretion or retention, for how could a metal whose urate is the most soluble of all urates yet cause retention of uric acid ?

There could be no doubt as to the great solubility of urate of lithia, and I could see no way out of the difficulty in that direction.

Thanks, however, to Dr. Neale's 'Digest,' I found a reference to the 'Lancet' twenty-eight years ago, which said that lithia was no use as a solvent of uric acid when taken by the mouth, and on looking up this reference I found the following statements ('Lancet,' ii, 1860, p. 185) :

"Mr. Ure does not recommend the internal use of this salt (carbonate of lithia) in the treatment of vesical calculus for the reason that it would undergo decomposition and precipitation when coming in contact with ammonio-phosphate of soda, and thereby be rendered 'inert.'"

FIG. 4.



Lithia Cit.  
gr. xxv in 3 doses.

Lithia Cit.  
gr. xx in 3 doses.

Lithia Cit.  
gr. xxvij in 3 doses.  
Sodii Phosph. 3j ter.

Sodii Phosph. 3j ter.  
No Lithia.

Lithia Cit.  
gr. xxvij in 3 doses.  
Sodii Phosph. 3j ter.

Given internally it forms a nearly insoluble triple phosphate with phosphate of soda, or with the triple phosphates of ammonia and soda, salts generally present in animal fluids. Lithia solutions differ in this respect from those of soda and potash."<sup>1</sup>

<sup>1</sup> Rose, 'Chemical Analysis,' p. 15.

The insoluble lithia phosphate above mentioned is, I believe, well known to chemists, and if we may take the chemical explanation as correct, it is easy to see that lithia taken internally not only never reaches the uric acid to form a soluble urate, but in forming an insoluble compound with phosphate of soda it practically removes from the blood one of the well-known solvents of uric acid ; and Fig. 4 shows, I think, that sodium phosphate taken in one-drachm doses three times a day, prevents lithia causing any retention, *i. e.* it supplies the place of the sodium phosphate removed by combination with the lithia, so that after all there is here no exception to the rule of solubility. I have now shown that three drugs, lead, iron, and lithia, when taken internally, cause retention of uric acid either by forming insoluble compounds with it or, as in the case of lithia, by removing one of its natural solvents ; and I now propose to look at what is known of their action in disease.

With regard to lithia I have very little to say. It was at one time much used, then fell into disuse, and has finally been brought forward again, in the treatment of uric acid disease, but there is by no means a unanimous opinion as to its value at the present day, and to quote<sup>1</sup> some recent writings, Dr. Yeo says of it, "For my own part I am disposed to think we are nowadays inclined to exaggerate the value of the lithium compounds as compared with those of potash and soda," and he quotes Ebstein, Sir W. Roberts, and Lecorché as doubting its efficacy, or preferring other alkalies, and he mentions that Ebstein asserts that chloride of lithium has no solvent action on uric acid.

I have above spoken several times of the action of dyspepsia, and it has some interesting connections in the causation of headache, and I believe also in that of epilepsy, gout, and other members of the group, of which I may here say a few words.

<sup>1</sup> 'Brit. Med. Journ,' i, 1888, p. 68.

In headache, the usual sequence is as follows :—There is a time (say seven to ten days) of good general health, active nutrition and bodily activity, with plus formation of uric acid and urea, and concomitant rise in acidity. As acidity rises, uric acid comes to be retained, and at the end of four or five days several grains may be regarded as stored up in the liver and spleen. Then come dyspepsia, gastro-intestinal catarrh, and hepatic congestion (and I am not by any means certain that this hepatic congestion and gastro-intestinal trouble may not be the direct result of the accumulation of uric acid in the liver and spleen). These quickly result in general diminution of absorption and nutritive changes, with lessened formation of uric acid and urea and a fall in acidity ; and lastly, as the result of this falling acidity, there comes a rush of the stored uric acid into the blood and the headache begins. Such a sequence may be seen to some extent in the figure that accompanies my paper on headache ('Transactions,' 1887), for there the urea drops from 561 grains to 363 grains in the four days that immediately precede the headache. Although acidity was not estimated in this instance there can be no doubt that it followed and shared in the fall of urea to a large extent. Such a sequence explains the periodicity of this kind of headache, and the way in which it<sup>1</sup> comes to occur every week or ten days for many years, varying only in degree with the corresponding variations in nutrition. It is also evident that any causes which affect digestion will influence the attack in one of the above ways ; while all causes of debility, by weakening the nerve-centre on which the uric acid acts, will render it more sensitive (the reverse of the action of bromides) and the attacks more frequent. A knowledge of these facts gives me almost complete power either to cause or cure this headache in myself and other sufferers. Further, I believe it will be found that dyspepsia bears a similar causative

<sup>1</sup> 'St. Bartholomew's Hospital Reports,' 1887.

relation to the attack of gout, epilepsy, and other members of the group, and that the conditions affecting the nerve-centres act in epilepsy just as in headache.

With regard to lead, I stand on very firm ground, for Sir A. Garrod has shown that it<sup>1</sup> diminishes the excretion of uric acid; and he has also shown that, given medicinally, it will precipitate attacks of gout; further he records the fact that in lead-poisoning the blood almost invariably contains uric acid.

Dr. Ross also speaks of chronic lead-poisoning<sup>2</sup> as a cause of epileptic convulsions, and Mr. Pepper, in the 'Lancet' of Nov. 5th, 1887, makes some interesting remarks on lead as a cause of epilepsy, which he regards as curious or even mysterious; but I cannot see that there is any mystery about it once we know that epilepsy may be (as I shall attempt to show farther on) due to uric acid, as we can then at once understand that the bad effects of iron and lead may result from the retention of uric acid they occasion.

Sir A. Garrod<sup>3</sup> has pointed out that iron given soon after an attack of gout will cause a relapse, and M. Brown-Séquard<sup>4</sup> says that iron does harm in many cases of epilepsy; it has also appeared to me to be harmful in some cases of headache due to uric acid,<sup>5</sup> so that I have several times been led to look for uric acid as a cause, from the fact that iron did harm.

With regard to salicylates in gout, though some have expressed doubts of<sup>6</sup> their value, and though Dr. Yeo speaks somewhat unfavorably of them, many of the opinions he quotes are strongly in their favour. Some, while acknowledging their value in the disease, refuse to give them on account of their depressant action, or some harm they may do to the kidneys.

<sup>1</sup> Garrod, 'Gout and Rheumatic Gout,' 3rd edit., p. 237, *et seq.*

<sup>2</sup> Ross, 'Diseases of the Nervous System,' vol. ii, p. 915.

<sup>3</sup> 'Brit. Med. Journ.,' i, 1883, p. 705.

<sup>4</sup> Quain's 'Dictionary of Medicine,' p. 450.

<sup>5</sup> "Uric Acid Headache," 'St. Bartholomew's Reports,' 1887.

<sup>6</sup> 'Brit. Med. Journ.,' i, 1888, p. 67.

From what I know of their action on the uric acid excretion I should not expect them to have a very prompt curative effect in acute gout, though, taken for some time, I should expect them to prevent attacks,<sup>1</sup> so that some unfavorable opinions may be due to their slow action in acute cases (see especially the opinion of Lecorché, quoted by Dr. Yeo, as to their power in prevention).

In explanation of the references made to epilepsy, I may say that I have shown in a paper in the 'Neurologisches Centralblatt' of March 1st, 1888, that some cases of epilepsy will yield uric acid reactions which are practically identical with those I have obtained in the case of headache,<sup>2</sup> so that if some headaches are due to uric acid some epileptic fits are also due to it, and the fits in these cases can probably, like the headaches, be prevented by treatment directed to the uric acid factor.

Just as the clinical relationship of headache to gout led me to examine the urine in headache, so the strong clinical relationship of epilepsy both to headache and gout<sup>3</sup> led me to expect, what I have now found, a similar uric acid reaction in each.

All epileptic fits are not due to uric acid any more than all headaches are; but I expect that it will eventually be shown that those cases of epilepsy which have a well-marked family history of gout, rheumatism, headache, or epilepsy will give uric acid reactions, and be cured by treatment directed to the uric acid factor, just as in the case of the corresponding headache; and it is in such cases of epilepsy that iron will do harm, that lead may be expected to precipitate attacks, and that salicylates will prevent them. And I believe that it may eventually be possible to show that salicylates do prevent the fits in such cases, as well or better than bromides.

If any of those who are interested in the pathology and

<sup>1</sup> 'Proc. Roy. Med. and Chir. Soc.,' Jan. 10th, 1888, N. S., vol. ii, p. 327.

<sup>2</sup> 'Med.-Chir. Trans.,' vol. lxxi.

<sup>3</sup> Paper on Headache, 'St. Barth. Hosp. Rep.,' 1887.

treatment of epilepsy should feel inclined to investigate the matter from this point of view, I would suggest the following method, which I now use myself:—Take a case of idiopathic epilepsy where, as in the case of the corresponding headaches, the fits have begun early in life and lasted for many years, where there is also a marked family history of gout, rheumatism, headache or epilepsy. The fits in such a case may be due to uric acid, and the point may be tested as follows:—If the fits are due to uric acid in the blood, drugs which prevent accumulation (retention) of uric acid will prevent the fits. Now, salicylates are, for the reasons I have given, probably the best of all drugs to prevent retention of uric acid; so that in the supposed case if salicylate of soda is given in doses sufficient to prevent any retention of uric acid (say ten grains for a child, or fifteen to twenty for an adult, three times a day) excess of uric acid in the blood will be almost impossible, and the fits will be completely absent so long as the salicylates are kept up and are absorbed. Where such immunity occurs, I should consider that it practically proves the fits to have been due to uric acid; but if there is any doubt a uric acid reaction taken during a fit will settle the question.<sup>1</sup>

In cases which have been tested as above by salicylates, or where uric acid reactions have been obtained, there is, I think, no doubt that the diet which I have found so useful in headaches due to uric acid, will in course of time afford what is practically complete immunity from fits, and cases cured by vegetable diet are not very rare (see my paper in 'St. Bartholomew's Hospital Reports' for 1887, p. 211).

And if salicylates can prevent these fits by preventing retention of uric acid, it is surely no very extraordinary thing that iron and lead, which cause retention, should occasionally produce fits as they have been observed to do.

And there is one further point about epilepsy, for a

<sup>1</sup> 'Neurologisches Centralblatt,' March 1st, 1888.



reference to which I am indebted to Sir Dyce Duckworth, viz., that Dr. Radcliffe<sup>1</sup> has noticed that by giving Potassium Iodide and Potassium Bicarbonate along with bromides he was able to give less bromide. Thus he says, "About the advantages of associating iodide of potassium and bicarbonate of potassium with ordinary bromides in the treatment of epilepsy, I cannot speak so positively as about the disadvantage of associating iron with them;" and further on he says, "I am inclined to think that less bromide will serve to produce a good result with this addition (iodide and bicarbonate) than without it. To this conclusion I am constantly led by the results of experience."

The explanation of this observation is quite simple, for I have shown in the 'Journal of Physiology' that alkalies facilitate the excretion of uric acid and prevent retention, and if alkalies are steadily given along with bromides there will never be any great accumulation of uric acid in the liver and spleen or a great excess of it in the blood; hence a smaller dose of bromide will serve to prevent the nerve-centres from reacting to the irritant. Dr. Radcliffe's remarks about iron, and also some other remarks in the same paper about diet in epilepsy, are of great interest from my point of view, and tend to confirm my explanation of its causation.

And I believe that very much of the same reasoning will apply to some important points in the etiology of gout into which there is no space to enter here; for instance, several writers on this subject speak of small temporary hindrances to uric acid excretion as causative of attacks. Thus Liebermeister, speaking of gout, says,<sup>2</sup> "In manchen Fällen scheint eine temporäre Behinderung der Ausscheidung durch die Nieren die Gelegenheitsursache des Anfalls zu sein," and a rise in acidity gives us just such a "temporäre Behinderung," though the fault is not in the kidneys themselves; but if salicylates are present in the circulation the rise in acidity will no

<sup>1</sup> 'Practitioner,' 1883, vol. xxx, p. 95.

<sup>2</sup> 'Vorlesungen über Special Pathology u. Therapie,' vol. iii, p. 38.

longer have power to cause retention of uric acid, and thus an attack of headache, epilepsy, or gout will be prevented.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 377.)



**ON THE RESULTS**  
**OF THE**  
**TREATMENT OF PULMONARY CONSUMPTION BY**  
**RESIDENCE AT HIGH ALTITUDES,**  
**AS EXEMPLIFIED BY AN ANALYSIS OF 141 CASES.**

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THE treatment of phthisis by residence at high altitudes, though extensively used by Drs. Archibald Smith, Guilbert, Lombard, Jourdanet, Brehmer and others, was first brought to the notice of this Society by Dr. Hermann Weber in an admirable paper to be found in vol. lii of the 'Transactions,' to which all workers on the effects of climates are much indebted, not only for the able discussion of the objections raised to Alpine climates, but also for the valuable record of seventeen cases of phthisis treated by residence at high altitudes in different parts of the world.

Since then a number of papers and works have appeared on the same subject contributed by Drs. Clifford Allbutt,

Spengler, McCall Anderson, Symes Thompson, Tucker Wise, Burney Yeo, Lindsay, and others, dealing for the most part with the climate of Davos and the Engadine and with the life of the invalid in these localities, but since the publication of Dr. Weber's cases we have had but few clinical illustrations of the effects of this climate on consumptives and still fewer statistics, the principal ones being Dr. Denison's<sup>1</sup> cases of consumption treated in Colorado.

Having made the acquaintance of Davos and the Engadine in 1862, I revisited these places in 1872, with the view of using them as summer and winter health resorts for English consumptives, but it was not until 1878 that the opening of an hotel specially arranged for English people, and the presence of an English-speaking doctor at Davos, enabled me to carry out my intentions to any extent. Since then St. Moritz, Wiesen and the Maloja have also been rendered available for the reception of patients in winter as well as in summer, and have been utilised by myself and others.

The object of this paper is to give a statistical account of 141 consumptive patients, who have lived for periods extending from months to years in high-lying localities, varying in altitude from 5000 to 9000 ft., either in Switzerland or in Colorado (Rocky Mountains), or in the South African Highlands, and to deduce from their histories and their results the kind of case most likely to benefit, and also that most likely to be injured, by this method of treatment; moreover, to draw up such practical rules as will render the climatic sojourn as useful and beneficial as possible.

I may remind the Fellows of the Society that this is the third series of statistics bearing on the climatic treatment of consumption offered by me to the profession, the first being a paper on the "Effects of Warm Climates in the Treatment of Pulmonary Consumption," as exemplified in 251 cases, contained in vol. lv of the

<sup>1</sup> 'Rocky Mountain Health Resorts.'

‘Medico-Chirurgical Transactions;’ the second, on the effects of the British South Coast and other home climates on 243 consumptives, was published in the Lettsomian Lectures for 1876, and it is hoped that a comparison between these and the present cases may afford the means of judging to some extent of the relative advantages and peculiarities of each form of climatic treatment. Of the present 141 cases 6 were published in the ‘Lancet’ of 1879, after my third visit to Davos, and 16 more were contained in a paper read at the International Medical Congress held in London in 1881, and included in the ‘Transactions’ of that meeting. Three cases more were contributed to vol. xvi of the ‘Clinical Society’s Transactions,’ making in all 25 which have been published before in some form or other. The remaining 116 are now presented to the Society, and together with the first 25, they form a number from which conclusions with reference to high-altitude treatment may safely be drawn. The cases are too numerous to give in full, and I have therefore tabulated them under the following headings:

Sex.

Age.

State at first visit.

Nature and extent of disease.

Length of illness.

Family predisposition.

Hæmoptysis.

Length of residence at high altitudes.

Results	{	General.
		Weight.
		Local.
		Chest circumference.

Remarks, including history up to date or death.

A specimen of such a table is shown.

No.	Name.	Sex.	Age.	Date of 1st visit.	Nature and extent of disease.	Length of illness.	Family predisposition.	Hæmoptysis.	Length of residence.
101	Mr. S.	M.	25	May 2, '85	Phth. I—III. Pigeon-breasted; softening of right upper lobe, and rheumatic fever frequently; mitral disease (regurgitant); syphilis.	About 4 years. Bacilli present	Mother d. phth.	5 oz.	St. Moritz 2 winters, Davos 1 summer
102	Mr. H.	M.	45	June 13, '85	Phth. I. Consolidation, of both apices; syphilis.	1 year	None	Large amount on several occasions	Wiesen 10 months.
103	Miss K.	F.	20	Sept. 22, '85	Phth. III. Cavity in upper left lobe; consolidation of lower lobe.	—	Mother d. phth.	None	Davos 4 months
104	Rev. J. C. M.	M.	28	Aug. 20, '85	Phth. II. Hæmorrhagic softening upper right lobe, with hæmorrhagic consolidation lower right.	6 weeks. Bacilli abundant.	Sister and paternal aunt and uncle d. phth.	10 oz. several times.	Davos 2 years, Pontresina 1 summer
105	Mrs. F. M.	F.	30	Aug. 15, '85	Phth. I. Tuberculisatation upper right lobe.	6 months	—	None	St. Moritz 6 months
106	Mr. M.	M.	22	Sept. 23, '85	Phth. III. Scattered cavities in in right lung.	18 mos. Bacilli few.	—	Some	St. Moritz 2 winters
107	Mr. C.	M.	20	Sept. 8, '85	Phth. I. Tuberculisatation, both upper lobes.	7 years. Bacilli present.	Father and brother d. phth.	None	St. Moritz 6 months
108	Mr. L.	M.	34	Aug. 28, '85	Phth. I. Tuberculisatation, both upper lobes.	9 mos., winter cough 10 years.	None	1 oz. several times	St. Moritz 6 months
109	Sir A. N.	M.	30	June 24, '85	Phth. I. Tuberculisatation of upper half of left lung.	3 years.	Paternal uncle d. phth.	None	St. Moritz 1 winter
110	Mr. C. E. N.	M.	26	July 23, '85	Phth. III. Contracting cavity in left lung; tuberculisatation in upper left lobe.	5 years. Bacilli abundant.	Mother d. phth.	None	Davos 6 months
111	Mr. R.	M.	34	Aug. 25, '85	Phth. I. Tuberculisatation of both upper lobes; fistula 6 months.	1 year. Bacilli present.	None	Some	Davos 2 years
112	Miss G.	F.	28	May 27, '85	Phth. I. Tuberculisatation extensive in right lung and in upper lobe of left.	3½ years. Bacilli abundant.	—	None	Davos 6 months
113	Mr. F.	M.	29	Oct. 2, '85	Phth. III. Cavity in right lung (middle lobe); tuberculisatation of upper left lobe; morbus cordis (aortic disease); occasional pyrexia.	1 year. Bacilli present.	None	Streaked expectoration	St. Moritz 2 winters
114	Mr. I.	M.	20	Mar. 15, '86	Phth. III. Cavity of left lung; syphilis.	1 year. Bacilli abundant.	Father, mother, and paternal uncles d. phth.	None	Davos 9 months
115	Miss S.	F.	27	Nov. 11, '85	Phth. I. Tuberculisatation of upper right lobe.	6 weeks. Bacilli present.	Brother d. acute phth., father d. phth.	None	Davos 2 winters
116	Rev. J. M.	M.	38	Oct. 9, '85	Phth. I—III. Softening and tuberculisatation both proceeding in left upper lobe.	4 months	None	None	Davos 2 winters

Result.				
General.	Weight.	Local.	Chest circumf.	Notes.
Greatly improved	Gain 7 lbs.	Advance of disease; cavity formed and began to retract; emphysema of right lung; hypertrophy of left lung.	—	1887.—During 1st winter cavity formed with much pyrexia, followed by an attack of rheumatic fever, with fresh endocarditis and pulmonary congestion. In 2nd winter improved till hæmoptysis, from which recovered, and now able to go about quietly.
Cure	Gain 5 lbs.	Arrest of disease, hypertrophy of both lungs	Increase	April, 1887.—Quite well and returning to practice as doctor. Complained of irritation of chest during stay at Wiesen, but steadily improved.
Deterioration, death.	0	Intestinal ulceration.	—	Feb., 1886.—Died of tubercular intestinal ulceration.
Deterioration	At first 7 lbs. gain	—	Slight increase	1887.—Improved 1st winter, but at Pontresina severe hæmoptysis, and again at Davos, Christmas, 1886, copious hæmoptysis with high temperature, and confined to bed for weeks. Sept., 1887, married and returned to England.
Great improvement	Gain 1 st.	Arrest of disease; emphysema of right lung and hypertrophy of left.	Increase	1887.—Passed last winter at Brighton, and has sustained her improvement.
Greatly improved	Gain	Partial arrest; emphysema.	Increase 1 inch	1887.—Improved greatly 1st winter; cavity almost closing and bacilli nearly disappearing from sputum. Returning to Ireland he relapsed, but improved again 2nd winter.
Cure	Stationary	Arrest of disease; extensive emphysema.	—	1887.—Returned from St. Moritz sufficiently well to resume medical studies. Continues in fair health.
Greatly improved	Gain 11 lbs.	Arrest of disease; development of emphysema and hypertrophy.	—	1886.—Returned to occupation in May.
Cure	Gain	Arrest of disease; disappearance of all signs except prolonged expiration above left scapula; lung hypertrophy (Holland).	Increase 1½ inches	1887.—In robust health ever since return from St. Moritz.
Deterioration; death subsequently.	Loss	Advance and extension.	—	Had attack of congestion at base of lungs with pleurisy, and reached home to die April 26th, 1886.
Great improvement	Gain	Improvement.	Increase	1887.—Though the area of disease was large, no cavity appeared to form, and the patient gradually improved enough to earn living at Davos as artist.
Great improvement	Gain	Partial arrest.	—	May, 1886.—Fairly well.
Deterioration, death.	Loss	Some extension; emphysema; some contraction of cavity.	—	1887.—During 1st winter felt effect of altitude on heart (palpitation, short breath), but soon better, and able to ascend hills; gained weight. Worse during summer in England, with occasional pyrexia. In 2nd winter at St. Moritz lost flesh through overwork as tutor. Had acute pleurisy at Ragaz on return home, and died May, '87.
Great improvement	Gain about 7 lbs.	Partial arrest; contraction of cavity; absorption of infiltration; lung hypertrophy.	Increase	1887.—Left Davos greatly improved for Les Avants. Still continues well.
Great improvement, cure.	No gain	Arrest of disease.	No increase	1887.—Very well since return to England.
Improved	Gain (little)	Advance of disease; cavity formed in left lung, and underwent some contraction; hypertrophy of right lung.	Some increase	1887.—Continues pretty well.



Two cases are furnished in full as examples.

CASE 1.—Miss K—, æt. 17, seen August 15th, 1884, with a history of cough, expectoration, night sweats, and loss of flesh since February last, the loss of weight amounting to seven pounds. Breath short on exertion. *The physical signs were, right side, flattening and crepitation in first intercostal space; left side, crepitation in first intercostal space.* Weight 8 st. 8 lbs.

Ordered cod-liver oil with hyophosphite of lime and counter-irritation to both sides of chest.

September 20th.—Cough worse, expectoration contains numerous tubercle bacilli. *Cavernous sounds audible in first and second spaces on right side and over the upper fourth posteriorly. On left side crepitation heard above the clavicle, as well as in first space.* Chest measurements at level of second rib, right fifteen and a half inches; left fifteen and a half inches. Ordered to winter at St. Moritz under the care of Dr. Holland.

Miss K— went to St. Moritz in October, and at first the cough and expectoration increased and she had some pyrexia, which yielded to antipyrin and good nursing. She steadily improved and was able to take moderate exercise, at first walks on the level, and then gentle ascents, and later on skating, but was forbidden tobogganing and lawn tennis. Cod-liver oil and tonics were persevered with. The pulse and respiration were 94 and 22 respectively on arrival at St. Moritz, and gradually fell to normal.

May 27th, 1885.—Has just returned and looks well browned and much fatter. Has gained nine pounds, weighing now 9 st. 3 lbs. Cough and expectoration slight. No bacilli found in sputum. Chest expands well in upper portions and appears to have increased specially in lower part.

Measurements at level of second rib : Right side sixteen and an half inches, left side fifteen and a half inches.

*Hyper-resonance over the whole chest. Curious band-box*

*sound on percussion under right clavicle, where indistinct cavernous sounds are still audible with some crepitation. On left side no crepitation, but harsh breathing with dry crackle everywhere. Ordered to pass a second winter in the Alps.*

June 3rd, 1886.—Wintered at Davos, gaining much strength. Was allowed by Dr. Ruedi to skate and toboggan, and often ascended the Schatz Alp, but took no cod-liver oil and lost some weight (about four pounds), nevertheless she appears wiry and looks the picture of health. No cough or expectoration. *No band-box sound, no cavernous sounds or crepitation anywhere, but some slight collapse under right clavicle; harsh breathing everywhere.*

In 1888 Miss K— continues quite well.

CASE 2.—Mr. C—, æt. 20, first seen September 8th, 1885. His father and one brother died of consumption. He had had a cough for six years, accompanied by loss of flesh and occasional night sweats, and had voyaged to South Australia and back without benefit.

*Dulness and flattening of the upper half right front chest, some dulness and crepitation in scapular region, dulness and flattening of upper third left front chest, with tubular sounds in first interspace. Cough very troublesome, and he states that he loses his voice, but the larynx on examination appears quite healthy. Expectoration contains tubercle bacilli.*

Chest measurements at level of third rib :—Right side seventeen inches, left side sixteen and a half inches.

Ordered to winter at St. Moritz under the care of Dr. Holland.

May 2nd, 1886.—Improved wonderfully at St. Moritz and soon lost cough and expectoration. Took a fair amount of exercise; weight about the same. Chest measurements show one inch increase in circumference. *The whole chest expands well, and there is no flattening anywhere, all dulness and crepitation have disappeared, and resonance is present everywhere, except over cardiac*

and hepatic regions. Prolonged expiration is heard under both clavicles, but otherwise the expiration is harsh throughout.

Mr. C— returned to his studies as medical student, has since completed his education, and still remains in excellent health (1888).

*Sex.*—The patients were 100 males and 41 females, belonging to the upper and middle classes of society, and therefore to a large degree secured from the reach of want or poverty.

*Age.*—Their ages at the time of commencing high-altitude treatment are classified in the subjoined table (Table I), which also contains the percentages of each sex and of the whole number.

The average age was for males 27·27 years, females 24·46.

TABLE I.—*Showing Ages of 141 Patients at the time of their Residence in High Altitudes.*

	Males.		Females.		Total.	
	No.	Percentage.	No.	Percentage.	No.	Percentage.
10 to 20 years .	14	14·	9	21·95	23	16·31
20 to 30 „ .	55	55·	24	58·53	79	56·02
30 to 40 „ .	22	22·	7	17·07	29	20·56
40 to 50 „ .	9	9·	1	—	10	7·09
Total .	100		41		141	

It will be seen that by far the greater number of both sexes were between twenty and thirty, only one fifth being between thirty and forty and one sixth under twenty. No less than 56 per cent. were in the twenty to thirty category, 55 per cent. of the males and 58½ per cent. of the females, and this fact and the average age show that the majority of these patients were

attacked rather earlier than the average consumptives of the upper classes, but were also brought under treatment earlier.

As appears in most statistics of the kind, more females than males were attacked before twenty, and more males than females after thirty.

*Date of first visit.*—This in most cases very shortly preceded the patient's leaving England, and as the first patients were sent in 1878, and in increasing numbers in succeeding years, a considerable period, amounting in some cases to eight or nine years, has elapsed since the first sojourn at high altitudes, and the question of the permanence of this treatment can be tested by the after-history.

*Length of illness.*—By this is meant the period intervening between the first symptoms of disease and the commencement of the mountain residence. This varied greatly in length; 56 of the 141 patients had been ill for less than one year, some for only two or three months, others for two, three, four, five, six, seven and eight years, and one had a history of ten years' illness and another a history of twenty years, but the greater number were sent to high altitudes comparatively early, and this is shown by the average length of illness, which for the males was twenty-four months, or two years, and for the females 19·31 months, or rather over one and a half years. The influence of this factor on the results, and also of the age of the patients, will be considered later on.

*Family predisposition.*—This was known to be present in 62 out of the 141, or in 44 per cent., and 23 cases were specially recommended for mountain treatment on account of the strong hereditary predisposition, these patients having lost one or both parents from consumption and also brothers or sisters, thus affording evidence of the existence of the disease in the same generation. Family predisposition was present in 32, or 32 per cent., of the males, and in 30, or 73 per cent., of the females, the

numbers confirming the former conclusions I arrived at :— that family predisposition is more potent among females and males.

*Hæmoptysis* had occurred to a lesser or greater extent in 61 cases, or in 43·26 per cent., a somewhat smaller percentage than that of the 1000 standard cases from the same classes contained in my “Duration of Phthisis” paper, where it was 57 per cent. Four of the cases belonged to the variety called hæmorrhagic phthisis, characterised by attacks of large recurrent hæmoptysis.

*History and nature of cases.*—Before describing the condition of these patients’ lungs, it may be stated that 3 had malformations of the thorax in the form of pigeon breast, 3 had the complications of syphilis with phthisis, 1 had fistula *in ano*, and 1 strumous disease of the hip, 1 had phthisis and emphysema, 4 had cardiac lesions (1 aortic regurgitant disease, and 3 mitral regurgitant), 2 of these being the result of rheumatic fever. Four patients, as stated above, were instances of hæmorrhagic phthisis, and one was a case of fibroid phthisis, but the result was so unfavorable that no more patients of this category were sent.

The rest of the patients were nearly all cases of chronic pulmonary phthisis, the symptoms and features of which it is hardly necessary to describe, but it is proposed to give a careful account of the state of the lungs, from which a good idea may be formed of the condition of these patients before leaving England. In most of these cases pyrexia was absent, but in 10 it had been present and had subsided shortly before starting. The degree of wasting was in no case very marked, though some patients had lost a stone in the course of their illness.

*State of the lungs.*—The chief conclusions from the examination of the chest will be found set forth in Table II, which, with a few explanatory additions, will exhibit the principal facts.

Of the 141 patients, 91, or 64½ per cent., were in the first stage, or that of tuberculisation ; 50, or 35½ per cent.,

TABLE II.—*Showing the Influence of High-altitude Climates on the Condition of the Lungs in 141 cases of Consumption.*

Stage.	No.	Percentage.	State of lungs before residence at high altitudes.	Result.								
				Arrest of disease.	Partial arrest.	Decrease of disease.	Stationary.	Advance of disease.	Advance and extension.	Extension.	Unknown.	Totals.
1st (tubercu- lisation)	91	64·53	37 had the <i>right</i> lung alone affected . . .	25	6	2	...	1	1	2	...	37
			23 had the <i>left</i> lung alone affected . . .	16	5	...	...	...	...	2	23	
			31 had <i>both</i> lungs affected	15	11	1	1	...	3	...	...	31
				56	22	3	1	1	4	2	2	91
2nd and 3rd (soften- ing and excava- tion)	50	35·46	13 had the <i>right</i> lung alone in 2nd or 3rd stage .	1	5	1	...	3	1	2	...	13
			6 had the <i>right</i> lung in 2nd or 3rd stage and the <i>left</i> in the 1st .	...	1	1	...	...	2	2	...	6
			15 had the <i>left</i> lung alone in the 2nd or 3rd stage	2	4	...	3	3	2	1	...	15
			15 had the <i>left</i> lung in 2nd or 3rd stage and the <i>right</i> in the 1st .	2	5	1	1	1	4	1	...	15
			1 had <i>both</i> lungs in the 2nd stage . . .	...	...	...	...	1	...	...	...	1
				5	15	3	4	8	9	6	...	50
Totals . . .				61	37	6	5	9	13	8	2	141

In 53 of the 141 patients both lungs were affected, *i. e.* in 37·58 per cent. In 88, or 62·41, one lung was attacked, the right in 50, or 35·46 per cent., and the left in 38, or in 27 per cent.

Results.		Per cent.	(Unknowns excluded.)	
1st stage 89 cases (unknowns excluded)	Arrest of disease . . .	56 = 63·	Improved	81 = 90·01
	Partial arrest . . .	22 = 24·73		
	Decrease of disease . . .	3		
	Stationary . . .	1	7·86	
	Advance of disease	Worse 7 = 7·86		
	Advance and extension Extension			
2nd and 3rd stages, 50 cases	Arrest . . .	5	Improved . . .	23 = 46·
	Partial arrest . . .	15		
	Decrease of disease . . .	3		
	Stationary . . .	4	4	
	Advance of disease . . .	8	Worse . . .	23 = 46·
	Advance and extension	9		
	Extension . . .	6		
Total	Arrest . . .	61 = 43·88	Improved	104 = 74·82
	Partial arrest . . .	37		
	Decrease of disease . . .	6		

	Per cent.	(Unknowns excluded.)
Stationary . . . . .	5	Per cent
Advance of disease . . . . .	9	
Advance and extension . . . . .	13	} Worse . . . 30=21.58
Extension . . . . .	8	
Unknown . . . . .	2	

were in the second and third stages, or those of softening and excavation. As regards unilateral and bilateral affections, 88, or 62 per cent., had one lung affected, the right in 50, or 35½ per cent., and the left in 38, or in 27 per cent., and 53, or 37½ per cent., had both lungs diseased. Taking the first stage, or the tuberculisation cases, we find that one lung was affected in 60 cases, or two thirds, both lungs in 31, or in one third. The right alone was attacked in 37 and the left alone in 23, thus confirming the conclusions of my former paper, that the right lung is more liable to tubercular attack than the left. The amount of tuberculisation varied greatly in these 91 cases, and may be classed in three categories.

- (1) Cases of incipient phthisis, where one or both apices were involved.
- (2) Cases where tuberculisation had affected the whole upper lobe of one or both lungs.
- (3) Cases where the consolidation was more extensive, and somewhat scattered in distribution, involving either portions of both lungs or else the whole of one lung.<sup>1</sup>

It was found that among the 91 first-stage cases, 75, or 82½ per cent., presented a considerable extent of consolidation, while only 16, or 17½ per cent., were instances of what may be called incipient phthisis, with only apical lesions, and attention is specially drawn to this feature.

<sup>1</sup> It is possible that this category may include a few cases of old contracted cavities of limited size, especially where the history of symptoms is of some length, but efforts have been made to eliminate these by careful auscultation and measurement of the chest, as well as, whenever possible, by sputum examination; should any remain they would not increase but diminish the favorable character of the cases.

Of the 50 cases of softening and excavation, 8 were instances of softening, where breaking down of the lung was proceeding, 7 of one apex, and 1 of both apices; and 42 of excavation, where as a rule this process was already accomplished. As these cases can with difficulty be separated, it has been thought advisable to place them together under the head of excavation. Of the whole number of 50 cases, 28 had excavation of one lung, and the opposite one free from disease, 21 had excavation of one lung and the opposite one tuberculised to a more or less extent.

In one case both lungs were undergoing softening. The right lung was the seat of softening or excavation in 19 and the left in 30, thus confirming the conclusion arrived at in my paper on the "Duration of Phthisis" in vol. liv of the 'Transactions' of this Society, that the left lung is more liable to excavation than the right.

The size of the cavities in these cases varied, but the excavation seldom extended beyond the upper lobe of one lung.

In many of the unilateral cases there was also tuberculisation of the lower lobe or lobes. In two patients one lung was the seat of two or more scattered cavities, and in two others the cavities were basic but still unmistakably tubercular, and in several of the bilateral cases where one lung was the seat of excavation, the middle or lower lobe of the opposite one was the seat of tuberculisation, indicating that the spread of disease had taken place from re-inhaled sputum into the bronchi supplying these lobes.

It may be noted that the greater part of the third-stage cases were cases of single cavity with limitation of the disease to one lung, the proportion being as 28 to 22, or nearly as 4 to 3, and this no doubt exercised a powerful influence on the future of these patients.

*Tubercle bacilli in the sputum.*—A large proportion of these patients were sent to the mountains before the methods of bacillus staining had come into use. Dr. Ruedi of Davos, Dr. Holland of St. Moritz and I have



examined the sputum in 54 cases. Tubercle bacilli were detected in 49 of these and were absent in 5; in 16 they were abundant.

In some of the patients a number of successive observations were made, but they are neither numerous nor complete enough to draw deductions from. In a certain number the bacilli disappeared from the sputum during mountain residence. No attempt will be made in this paper to determine the exact influence of the mountain atmosphere on the parasite, but a simple statement is made of the result of the testings.

*Medicine and diet.*—These patients were treated with cod-liver oil and various tonics, and with such other measures as were required for urgent symptoms or intercurrent affections, but the cod-liver oil was not so resolutely persevered with, as in my home climate cases, partly, I have no doubt, because most of the patients felt, after a time, so well, as to deem themselves independent of medicine.

The diet was the usual one of Swiss hotels of three meals a day with meat twice and sometimes thrice daily, and in addition the patients generally took one pint of milk a day. Stimulants in the form of claret and Vältelliner wine were generally made use of.

#### *Length of Residence at High Altitudes.*

The tables show that collectively 141 patients spent 160 years at high altitudes, giving an average of 1 year 1·67 months per patient.

We also find that of the 141 patients 81 passed eighty-five years eleven months at Davos, giving an average of a little over one year for each at that health station; that 59 spent forty years six months at St. Moritz, giving an average of 8·23 months, and that 2 patients passed three and ten months respectively at Wiesen; that 4 patients resided during nine, five and a half, two and a quarter, and half a year respectively alternately at Davos and in

the Engadine, practically never quitting high altitudes, and that 4 patients lived at the Maloja, 3 for six months each and 1 for three months. Turning to America, we find that 2 consumptives resided in Colorado, each at altitudes varying from 6000 to 9000 feet, for periods of three years. Lastly, we see that 2 patients tried life in the South African Highlands at elevations of from 4000 to 6000 feet, one for six years, and the other for two years.

Now, this average length of residence at high altitudes of 1 year 1½ months is somewhat deceptive, and appears to be largely due to the presence in the statistics of a comparatively limited number of patients who resided for several years in the mountains, some four, five, six, and nine years respectively, remaining there often after arrest of disease had taken place, for reasons of preference or precaution. If we deduct the patients who spent one year or upwards in these regions, we find that the remaining 92 patients passed 49·9 years, giving an average of 6·22 months, *i. e.* practically equal to one winter.

It will be noted that 19 spent from two to three and a half months only, and these were summer months and not winter.

The majority of the patients passed the winter months at Davos or in the Engadine, and a smaller number extended their stay into the summer, leaving the heights only during the snow-melting, and some passed a second winter and even a third. Before proceeding to the results accruing from this treatment, let us briefly allude to some of the climatic features of the above-mentioned health resorts, and consider the kind of life the invalids passed during their stay in them.

It will be obvious that in our list of stations there is considerable difference of climates, all agreeing in the peculiarity of elevation above the sea level. Colorado has a higher mean temperature than the Grisons, and the South African Highlands of the Orange Free State and Transvaal have a still higher record. As the majority of the patients now under consideration frequented Davos

and the Engadine, it may not be out of place to say a few words on the climate of the Alpine high-altitude stations generally, which will apply equally to Davos (5200 feet), Wiesen (4770 feet), St. Moritz (6090 feet), and Maloja (5941 feet). As the most typical, we will select the winter climate, and it is moreover at this season that, having shaken off the impurities due to dust and tourists, this region assumes its white robe of snow and settles down into the wholesome life of a well-regulated health resort, where the food, habits, and the form and amount of exercise of the invalids are under the strict supervision of the medical attendant. The carrying out of this *régime* is facilitated by the comparative isolation of the valleys, and the absence of many of the attractions of the southern resorts. The patients repair to the Grisons in October, generally before the large snowfall takes place, in order to arrive at their destination on wheels and not in sleighs. They then spend five to six months at the chosen station, being in the open air as much as possible during the hours of sunshine, and retire to their hotels at sunset only. The form of exercise to be used is limited by the strength of the patient and arranged on a graduated scale, commencing with skating and walking on the level and up moderate heights, or advancing to tobogganing—of which, not the swift descent, but the slow and gradual ascent of the snow slope, dragging the toboggan, is the beneficial part—and in time arriving at mountain climbing and short walking tours.

During the first six weeks or two months the pulse and respiration are quickened, and the patient often complains of shortness of breath, but gradually expansion of the lungs and the consequent widening of the chest takes place, which ladies recognise as necessitating certain adjustments of stays and dresses, and both pulse- and respiration-rate fall to normal and sometimes even below. It is not uncommon for fresh arrivals, and especially elderly people, to complain of sleeplessness, but this generally passes off in about ten days.

The climate is cold, with sufficient sunshine to admit of sitting out of doors. In the sunshine the heat is as great as—sometimes greater than—on the shores of the Mediterranean, the solar radiation thermometer showing  $130^{\circ}$  F., while in the shade it almost always freezes, and the mean temperature for the winter months varies from  $26^{\circ}$  F. to  $30^{\circ}$  F. At night the temperature falls to far below the freezing point and sometimes to  $24^{\circ}$  C.;<sup>1</sup> and yet patients sleep with windows open with impunity.

The range of temperature between the mean maximum in the shade by day and the mean maximum in the shade by night amounts to  $22^{\circ}$  F. As might be expected with so cold an atmosphere, it is very dry; it is also remarkably still.

When snow-melting commences, which is generally at the end of March or the beginning of April, the sloppy condition of the roads and paths renders a descent to a lower level desirable; though, owing to rapid evaporation, the hygrometer indicates only a slight increase of humidity; and the patients then move to places of an altitude of from 2000 to 3000 feet, such as Thusis, Ragaz or Seewis, Gais, or Weissbad, and afterwards either return to the mountains, or travel to England, as the case may be. In some years, when the snowfall is not heavy, the melting is not sufficient to require patients to leave, and they remain at mountain stations continuously.

The characteristic elements of Alpine climates seem to be—

1. Diminished barometric pressure and consequent rarefaction of the atmosphere.

2. Diathermancy of the air, or the increased facility by which the sun's rays are transmitted through the attenuated air. This, according to Dr. Denison, causes an increase in the difference between the sun and shade temperatures of  $1^{\circ}$  F. for every rise of 235 feet.

3. Dryness of the air, owing chiefly to the low tempe-

<sup>1</sup> See 'Davoser Blätter,' Jan. 14th, 1888.

rature and consequent comparative absence of fog and mist.

4. Absence of strong currents.

5. Aseptic character of the air and freedom from organic germs.

The climate of Colorado has most of the above attributes, and in addition to this, owing to the higher mean temperature, the snow-melting does not take place on such a scale as to render a descent to lower levels necessary ; and consequently patients remain there all the year round, camping out in the summer, and they are also less limited in their walks and excursions in winter. The same may still more strongly be urged on behalf of the South African Highlands, and here the life in the open air and on the *trek*, sleeping by night in waggons and shooting game by day, is exceedingly beneficial and has been the means of arresting disease in many patients, and one of these is tabulated among the present 141 cases.

*General results.*—We will now consider the results of the high-altitude treatment on these 141 consumptives, which are described under the headings “general” and “local,” *general* referring to the general health, vigour and weight, and to the main symptoms, including cough and expectoration, and *local* including only the results of the examination of the lungs.

Under the head of general results are five categories :  
1. Cured. 2. Greatly improved. 3. Improved. 4. Stationary. 5. Deteriorated or Worse.

By cured I mean complete restoration to health, with the disappearance of all symptoms of disease.

			Percentages.
Cured	58	...	41·13
Greatly improved	42	...	29·78
Improved	16	...	11·34
Stationary	1	...	
Worse	24	...	17·02

Five patients died during the mountain residence, and 17 more on their return to their homes, after intervals of from a few weeks to nearly three years.

Most of the deaths are included in the deteriorated class, but the single stationary case also deteriorated in the winter following, and gradually becoming worse died two and a half years after leaving Davos; while 3 of those who improved at high altitudes relapsed afterwards and died at intervals of from two to three years after their mountain treatment. One of these, a young lady of exceedingly consumptive family history, with disease at the left apex, who passed one winter at Davos, lost apparently all symptoms and nearly all signs of disease in her chest. She then married a gentleman far advanced in consumption and went to Colorado and New Mexico, where he succumbed. During his illness he was nursed entirely by her. She returned to England in the following spring with cavities in both lungs and with intestinal ulceration, and she died in the autumn.

The following is a list of the causes of death :

4	died of hæmoptysis.
3	„ intestinal ulceration.
1	„ embolism of the pulmonary artery.
2	„ severe dyspnœa following fibrosis and dropsy.
1	„ acute pleurisy.
11	„ of the usual waste and exhaustion of con-
—	sumption.
22 <sup>1</sup>	

This is a list of the ascertained deaths among the 141, and we have news of all the rest, with very few exceptions, up to present date, so it is only possible that one or two deaths could have occurred among those few exceptions who have been lost sight of.

This mortality of 22,<sup>1</sup> or 15·60 per cent., spread as it is over ten years, is satisfactory.

<sup>1</sup> Three patients have died since the reading of this paper, raising the number of deaths from nineteen to twenty-two.

When we turn to the "cured," the result of 58, or 41·13 per cent., attaining to this condition is remarkable. The number of "greatly improved" reaches 40, or 28 per cent., and if we bracket "cured," "greatly improved," and "improved" together, we arrive at a total of 116 improved, or no less than 82·25 per cent., a striking figure considering that 50 cases of softening and excavation are included in the list. The "worse" class number 24, or 17 per cent.

The general appearance of the "improved" class is that of bronzed vigorous individuals, with remarkable walking powers, and among the cures it is not uncommon to find men who can walk thirty, and women who can walk fifteen and twenty miles a day, and can ascend snow-peaks. The appearance of the whole of the "improved" class leaves nothing to be desired, though of course they will not all stand great tests of endurance.

*Weights.*—The weight of 131 patients was registered, with the following result:

			Percentages.
Gained weight	96	...	73·28
Stationary	14	...	10·68
Lost weight	21	...	16·03

This is fair, considering that the diet, though good, is not so nutritious as the English food, and the amount of exercise taken is very large, consequently the tissue waste must be great.

Dr. Holland, of St. Moritz, drew my attention, during my last visit in winter to that health resort, to the fact that some cases of chronic phthisis when there is no fever, when the cough and expectoration are diminishing, and when the patients are apparently going on well, still lose flesh, even to the extent of four or eight pounds, and he attributed it partly to the increased amount of exercise taken and partly to the very dry atmosphere which surrounds them. The dryness of the air has been shown by Mr. Waters to be almost excessive.

Before considering in detail the local results in these cases, it may be worth while to draw attention to the principal changes in the thorax and lungs during residence at high altitudes, which have been noted by myself and others, and which have been described by me at some length elsewhere.<sup>1</sup>

Briefly they are these :

1. The thorax increases in circumference to the extent of from one to three inches, such increase taking place in the antero-posterior, the lateral, and sometimes in the vertical directions, and at various levels, but more commonly in the upper portions than in the lower. With this is combined increase in respiratory power, as shown by the depth of inspiration and by spirometrical measurements.

2. Greater resonance on percussion. This is noticeable in the chests of healthy persons, but far more so in consumptives, where there have been distinct areas of dulness. The diminution of dulness is almost invariable, and the deepening of the percussion-note, even in the neighbourhood of extensive consolidation, is so marked that one can generally tell from a chest examination if a patient has or has not recently come from high altitudes.

3. The harshness of the inspiratory sound, audible from the apices to the bases of the lungs. This is invariable over the healthy portions of lung tissue, and in the cases of arrested first-stage phthisis it replaces the areas of tubular sound, scattered crepitation, and bronchophony.

4. The substitution of dry for moist sounds, which has long been observed by the medical men practising at high altitudes. This is to be noted not only in the case of cavities, but also where formerly scattered humid crepitation was heard, and apparently no cavity has formed.

The widening of the thorax, the increased force and depth of inspiration, the deepening in the percussion-note,

<sup>1</sup> 'Transactions of the International Medical Congress, held in London, 1881.'



seem to be due to hypertrophy of the healthy lung tissue, *i. e.* to such further development as takes place in any organ from fuller and more complete physiological activity, and this is confirmed by these changes being accompanied by a reduction in the respiration-rate, which, unless there be great pulmonary destruction, occurs generally within the second or third months of high-altitude residence.

When genuine hypertrophy cannot take place, by reason of the diseased condition of the pulmonary tissue, and its lack of proper blood supply, vesicular emphysema results, this being formed chiefly around tubercular nodules, caseous masses, and in the neighbourhood of contracting cavities, whose presence it often masks. The emphysema appears to be the chronic local vesicular form well described by Sir William Jenner.

It is not impossible that both hypertrophy and emphysema may coexist in the same lung, the upper lobe being the seat of scattered consolidations surrounded by emphysematous vesicles, and the lower lobe free from disease and having undergone genuine hypertrophy.

After this explanatory statement we will now consider the *local* results of high-altitude treatment, first defining the terms "arrest," "partial arrest," "decrease of disease," "stationary," "advance of disease," "advance and extension," and "extension," under which headings the results are arranged.

*Arrest* signifies in the first-stage patients disappearance of all physical signs of disease. This may be so complete that the medical examiner, without referring to his notes, will not know which lung had been attacked.

In softening and excavation cases arrest means entire disappearance of cavernous sounds, and even of signs of consolidation, and nothing to be detected beyond deficiency of expansion, some harsh breathing over the whole side, and tubular breathing or prolonged expiration above the scapula. In some cases not even these signs were present. In other contracted cavity cases there was a

certain degree of immobility of the side and flattening of the chest wall, combined with hyper-resonance and prolonged expiration sound.

*Partial arrest* means that though there is evidence of arrest of the disease, still some physical signs remain, indicating generally limited consolidation, or possibly a contracted cavity.

*Decrease of disease* signifies simply a diminution of the physical signs, either in extent of area or in intensity.

*Advance of disease* means the process of softening and excavation of tubercular consolidations.

*Extension of disease* the spread of tuberculisation in one or both lungs.

*Advance and extension* a combination of these two processes.

*Stationary* indicates a condition in which there is no diminution in the original lesions, though it does not preclude hypertrophy of portions of the lung or lungs not already attacked.

The *local* results are fairly shown in Table II, to which a summary is appended. We see here that out of the whole number, in 61, or nearly 44 per cent., arrest of the disease took place, in 37, or 26½ per cent., there was partial arrest, and in 6 decrease of disease; that in 5 the disease was stationary, in 8 it advanced, in 9 it advanced and extended, and in 8 it extended. Bracketing "arrest," "partial arrest," and "decrease of disease," we arrive at a total of 104 improved, or 74·82 per cent., not so high a percentage as in the *general* list, which was 82, but still a very favorable one. Throwing together the "advance," "advance and extension," and "extension," we arrive at 30, or 21½ per cent., of "worse." We should note, too, the small number of the stationary class, only 5, or 3·59 per cent., which indicates that the climatic treatment was by no means negative in its result, but acted powerfully in one or other direction.

Most instructive is the study of the results of the different stages of the disease, for we find that, excluding

2 "unknowns," of 89 in the first stage arrest of disease took place in 56, or 63 per cent., partial arrest in 22, or 24.73 per cent., decrease of disease in 3, advance in 1, advance and extension in 4, extension in 2; and a stationary condition was maintained in 1, or, in other words, arrest or improvement took place in 81 out of the 89, or in 91.01 per cent., and deterioration ("worse") in 7 only, or in 7.86 per cent.

With reference to the question of one or both lungs being attacked, and its bearing on the prognosis, we find that the 58 cases (unknowns deducted) of unilateral affection gave 41 cases, or 70.68 per cent. of arrest, 11 of partial arrest (18.96 per cent.), 2 of decrease of disease, and 4 of "advance," "advance and extension," and "extension," *i. e.* 44 or 92.11 per cent. of "improved," and only 4 deteriorations, or 6.89 per cent., whereas of 31 cases of double affection 15 were "arrests" (48.38 per cent.), 11 (35.48 per cent.) were "partial arrests," 1 showed "decrease," against 3 "advance" of disease, "advance and extension," and "extension;" also 1 stationary, making 28 cures and improved (90.32 per cent.) and only 3 deteriorations (9 per cent.). The difference in the first-stage class between the total numbers of the improved in the unilateral and bilateral cases is not very striking, indicating that great progress towards arrest was made, even in cases of double affection, but when we compare the relative numbers in the "arrest" class we find the true significance of the double affection, for these show us that whereas this latter class counted 48 per cent. of "arrests," the single lung cases counted 70.68 per cent.

As to the relative chances of the two lungs in the race for recovery, it would appear that the left lung has rather the best of it, as out of 21 left-sided cases, "unknowns" having been excluded, all were improved (16 arrests and 5 partial arrests), whereas among the right-sided ones 37, or 89 per cent., had improved, including 25 arrests (67.56 per cent.).

Passing to the second- and third-stage patients we see that out of 50 cases 5 were instances of arrest, 15 of partial arrest, and 3 of decrease of disease. In 8 the disease advanced, in 9 it advanced and extended, and in 6 it extended; in 4 it remained stationary. This gives 23 cases (46 per cent.) of improved, including 5 arrests, with 23 instances of deterioration, *i. e.* equal numbers, or nearly half of the total number, either gained or lost ground. This is a good result when we remember the large number (22) of double affection cases, but it points very decidedly to the different expectations to be held out in cases of consolidation and of excavation respectively, for be it remembered that in the first stage the relative numbers were: improved 91·01 per cent., and deteriorated 7·86 per cent.

Further analysing the returns of the cavity cases we see that the 28 instances of single cavity yielded better results than where both lungs were affected, the single cavities giving three cases of "arrest," 9 of "partial arrest," and 1 of decrease of disease, against 12 "deteriorations" and 3 "stationary," or, putting it into percentages, "improved" 46·42 per cent., "worse" 42·8 per cent., whereas the double affection cases yielded 2 arrests, 6 partial arrests, and 2 decrease of disease against 11 deteriorations and 1 stationary, or "improved" 45·45 per cent., "worse" 50 per cent., a result which gives the advantage to the single cavity cases.

With regard to the relative prognosis of the two lungs after excavation has taken place these cases furnish the following: 19 *right* "single" and "double affection" cases yielded 1 arrest, 6 partial arrests, 1 decrease of disease, *i. e.* 9 improved (nearly half) against 10 deteriorations.

The 30 *left* "single" and "double affections" of cavity cases give 4 arrests, 9 partial arrests, 1 decrease of disease, *i. e.* 14 improved against 12 deteriorations, 4 being stationary. Placing it in percentages:

	Improved.	Worse.	Stationary.
Right lung cavities	47·37	52·53	
Left     ,,     ,,	46·67	40	13

The difference of result here shown is not very marked, but it indicates that the left lung cases showed less tendency to change, either for the better or the worse, than the right.

To sum up the local results :

In the 141 consumptives improvement, greater or less, took place in 74·82 per cent., arrest of disease in nearly 44 per cent., and deterioration in 21½ per cent.

Among the first-stage cases there was improvement in 91 per cent. and arrest of disease in 63 per cent., with deterioration in 7½ per cent.

Cases of unilateral first-stage disease yielded 92 per cent. "improved," including 70½ per cent. of "arrests," whereas cases of bilateral affection of the first stage give 87 per cent. "improved" with 48½ per cent. of "arrests."

In the second- and third-stage cases there was improvement to a greater or less extent in 46 per cent., arrests in 10 per cent., and deterioration in 46 per cent.

Single cavity cases gave better results than cavity cases with the opposite lung involved; and left lung cavities showed less tendency to change, either for better or worse, than the right ones.<sup>1</sup>

<sup>1</sup> It will be of interest here to refer to Dr. Denison's statistics of 202 consumptives residing in Colorado at altitudes of 5000 feet and upwards. They consisted of 148 males and 54 females, and had an average history of two years' illness before arriving at Colorado; 37 per cent. were in the first stage and 63 per cent. in the second and third stages; 56 per cent. had both lungs affected. The average stay in Colorado was one year and nine months.

As compared with my cases these patients included more second- and third-stage cases and examples of double affection, but their average stay at high altitudes was far longer; 69 per cent. were "much improved" and "slightly improved;" 12 per cent. showed "favorable resistance," which equals, I suppose, "stationary," and 28 per cent. showed "extension or advance of disease;" 20 per cent. of the patients died.

A further study of the details shows (1) the great number of the improved to be among the first-stage cases; (2) that the number of stationary cases in the total was small; (3) that the larger proportion of "deteriorated" came

*Chest measurements.*—These were carried out with Hare's measuring tape at various levels, those generally selected being that of the third rib, the mammary level, and the level of the ensiform cartilage. In some cases calipers measurements were used, and in a good many tracings were made by the aid of the cyrtometer.

It is not proposed to give the whole details of this work as the subject was discussed in reference to a smaller number of patients by me elsewhere,<sup>1</sup> and the present observations confirm the former ones.

Measurements were taken in 86 of the 141 patients, and in 83, or in 96½ per cent., considerable increase in chest circumference was found to have taken place. In 3 no increase occurred. In one of these there had been pleurisy on both sides of the chest, and there were probably extensive adhesions with some fibrosis; in another both lungs were affected, and a cavity existed in the left lower lobe during the mountain residence. Another cavity then formed in the upper right lobe, and some contraction of one side of the chest took place, but marked signs of emphysema were detected on the opposite side.

In these two cases the measures on return were identical with those taken before departure. In the third case the measures were not as accurately taken as I could desire. With these three exceptions expansion of the thorax was noted in all the cases where measurements were taken. The largest amount recorded was three and a half inches at the third rib level.

#### *Influence of Sex.*

Did the males reap more benefit than the females from the mountain climate? The natural inference would be from the cavity cases. Dr. Denison's statistics, though wanting in many details, such as the record of the extent of lung mischief—and not so favorable in results as mine—support the conclusions arrived at above, and are the more interesting as furnishing large evidence of the same healing influence being turned to account in the Rocky Mountains as has been done in the Alps.

<sup>1</sup> 'Trans. International Medical Congress, London, 1881.'

that the sex, which was able to take the largest amount of outdoor exercise, would profit most.

On comparing the extent of disease present in the patients of each sex, I cannot detect any material difference; 35 per cent. of the males were in the excavation stage, and  $36\frac{1}{2}$  per cent. of the females; 37 per cent. of the males had both lungs affected, and 31·70 per cent. of the females.

Turning to the general results we find that males counted 43 "cured," or 43 per cent., and 83 "improved," to 17, or 41·46 per cent., "cured" among the females, with 33, or 80·43 per cent., "improved." The results of the two sexes may be considered even, the slight difference of 3 per cent. being explained by the slightly larger number of cavity cases among the women.

*Influence of Age at Time of commencing Alpine Residence.*

A reference to Table I shows us that the great majority of the patients were between twenty and thirty when they commenced high-altitude treatment, only one fifth being under twenty, and about one fourth over thirty.

The average has been taken of each sex in each category of the general results with the following conclusions:

	Males.	Females.
Average age of "cured"	23·8	24·9
„ of "greatly improved"		
„ and "improved"	29·7	25·4
„ of "deteriorated"	30	19·6

The range of age among the males was somewhat wider than among the females, and this may account for the difference between the ages of the "cured" and "improved" among the former. The contrast of the ages in the "deteriorated" is most striking, and leads us naturally to the conclusion that the climate does not so well suit females under twenty or males over thirty, in fact it would appear that the younger a man is the more likely

is his system to benefit by the expanding and bracing influence of mountain climates, and on the other hand, for a woman to be similarly benefited, her frame must have attained to that standard of development and circulatory power which enables her system to withstand the great cold.

*Influence of Family Predisposition on the Results of Mountain Treatment.*

Did the cases in which family predisposition was present fare worse than the rest? In 62 this feature was traced, and in 23 it was strongly present. These 62 yielded 28 cures (including 10 of the most marked examples of predisposition), 17 greatly improved, 6 improved, 1 stationary, and 10 deteriorated. Now, placing these into percentages and comparing them with the total results, we get:

	Whole number of cases.	Cases with family predisposition.
	Per. cent.	Per cent.
Cured	82·25	82·25
Greatly improved		
Improved		
Deteriorated	17·02	16·12

Thus it appears that this element exercised no check on the favorable progress of the case, and that these patients did as well as the rest; and as no less than 17 of the 23 cases of marked predisposition improved, we may fairly claim the mountain climate as a powerful antidote to this terrible and often fatal influence in cases of phthisis.

*Influence of Length of Illness.*

As we have stated before, these patients gave a history of symptoms varying from a few months to several years before commencing mountain residence, and an attempt has been made in the annexed table (Table III) to determine how far the length of the illness influenced the results. The average length of history was for the males two years, and for the females 19·31 months. The patients are divided into



four categories according to the duration of the symptoms, and it will be noted that more than half these were of less than one year's duration. We see that the number of deteriorated (worse) steadily increases with the long standing of the disease.

TABLE III.—*Showing Influence of Duration of Illness on the Result of High Altitudes in Treatment.*

No. of patients.	Duration of illness.	Cured.	Greatly improved and improved.	Stationary.	Deteriorated.	Cured. Per cent.	Improved. Per cent.	Deteriorated. Per cent.
42	6 months and under	21	16	—	5	50·00	38·09	11·90
40	6 to 12 months (inclusive)	15	18	1	6	37·50	45·00	15·00
19	From 1 to 2 years „	8	7	—	4	42·10	36·89	21·05
40	Periods exceeding 2 years	14	17	—	9	35·	42·5	22·5

The patients with a history of six months and under, show 11·9 per cent. of worse, those with histories exceeding two years show 22½ per cent., nearly double the former.

Those in the first category claim 50 per cent. of cures, against 35 per cent. in the last.

The improved class does not present such a contrast, and it is evident that the cases of older standing, while not capable of counting so many complete cures, are able to display considerable improvement.

The table warrants us in concluding that mountain climates are most effective in arresting phthisis where the disease is of recent standing,<sup>1</sup> but they are also beneficial in cases where the disease has existed for a longer time.

#### *Influence of Mountain Treatment on Hæmoptysis.*

Sixty-one of these patients had had hæmoptysis previous to their residence in high altitudes; only 6 spat blood

<sup>1</sup> This agrees with Dr. Denison's conclusion—"The resort to a high-altitude climate by a consumptive gives a proportionately better result the earlier it is undertaken."

during their mountain sojourn ; in one case it was fatal and in 3 others, profuse. In 2 patients hæmoptysis occurred while descending from Davos to lower levels. Four of the patients were examples of hæmorrhagic phthisis, and 2 of these during their stay in the mountains were quite exempt from hæmoptysis ; the other 2 had attacks, but less severe and less frequent than they had had formerly at low levels.<sup>1</sup> The above evidence points to hæmoptysis being less common at high altitudes than at low levels, but the climate does not entirely preclude its occurrence.

We believe the facts contained in this paper warrant the following conclusions :

1. That prolonged residence at high altitudes produces great improvement in the majority of consumptive patients, and complete arrest of the disease in a considerable proportion, such arrests being of a more or less permanent character.

2. That in order to secure these advantages patients must be free from pyrexia and all acute symptoms, and must possess sufficient lung surface to adequately carry on the process of respiration in the rarefied atmosphere.

3. That in the cases of unilateral tubercular consolidation more or less extensive improvement occurred in 92 per cent. and complete arrest in 70½ per cent., and that the left lung showed greater tendency towards such arrest of disease than the right lung.

4. That in cases of bilateral tubercular consolidation there was improvement in 87·09 per cent. and arrest in 48·38 per cent.

5. That patients in the stage of softening and excavation (the cavity or cavities being limited to one lung)

<sup>1</sup> Dr. Denison holds "that the advantages of high altitudes are pre-eminently for hæmorrhagic cases in the first stage, while hæmorrhagic cases with excavations, especially if the bleeding has been recent and softening is in progress, should be interdicted from high elevations." I have known, however, cavity cases, with formerly large and recurrent hæmoptysis, perfectly recover at high altitudes, and remain free from hæmorrhage.

showed improvement in about half their whole number and deterioration in the same proportion, the arrests not exceeding 10 per cent.

6. That single cavity cases, where the opposite lung was free from disease, reaped more advantage than cavity cases with double affection, and that the left-sided cavities showed less tendency to extension or advance of disease than the right-sided ones.

7. That the influence of the climate seems to promote a change in the lungs, whether of a curative or destructive character, and to oppose quiescence.

8. That residence at high altitudes causes enlargement of the thorax, hypertrophy of the healthy lung tissue, and the development of pulmonary emphysema around the tubercular lesions, and that this expansion of the chest is accompanied by a diminution of the pulse- and respiration-rate.

9. That it is probable that the arrest of consumptive disease is partly owing to the pressure exercised on the tubercular masses by the increasing bulk of the surrounding tissue, which by emptying the blood-vessels of the part produces caseation and cretification of the tubercle.

10. That the above local changes are accompanied by general improvement, shown in the cessation of all symptoms and the gain of weight, colour, muscular, respiratory, and circulatory power.

11. That consumptives of both sexes benefit equally by mountain residence, but that the age of the patient exercises considerable influence on the result, females under twenty and males over forty reaping least advantage from such residence. Between 20 and 30 both sexes showed great improvement.

12. That the high-altitude treatment seemed to be specially adapted in cases where hereditary and family predisposition was present, and to exercise a distinctly counteracting influence over the development of the disease.

13. That the climate is beneficial in cases of hæmor-

rhagic phthisis, and that hæmoptysis is of rare occurrence at the mountain stations.

14. That mountain climates are most effective in arresting phthisis when the disease is of recent date, but they are also beneficial in cases of longer standing.

15. That the special effects of high-altitude residence on the healthy and sick are common to all mountain ranges of elevations of from 5000 feet and upwards, and have been observed in the Alps, the Rocky Mountains, the Andes, the Himalayas, and the South African Highlands.

16. That to ensure the full advantages of high-altitude residence a period of at least six months is necessary, and in cases of long standing and of extensive lesions one or two years are often requisite to produce arrest of the disease. That nevertheless arrest of the disease sometimes takes place after so short a period as two or three months' residence, and was noted in 6 per cent. of the patients.

17. That in addition to the above examples, mountain climates are beneficial in (1) cases of imperfect thoracic and pulmonary development, (2) in chronic pneumonia without bronchiectasis, (3) chronic pleurisy, where the lung does not expand after removal of the fluid, (4) spasmodic asthma without much emphysema, (5) and in anæmia.

18. That they are contra-indicated in the following conditions: (1) Phthisis with double cavities, with or without pyrexia. (2) Cases of phthisis where the pulmonary area at low levels hardly suffices for respiratory purposes. (3) Catarrhal phthisis and laryngeal phthisis. (4) Erethric phthisis, or phthisis where there is great irritability of the nervous system. (5) Emphysema. (6) Chronic bronchitis and bronchiectasis. (7) Diseases of the heart and great vessels. (8) Affections of the brain and spinal cord and conditions of hyper-sensibility of the nervous system; and (9) where the patients are of advanced age, and where they are too feeble to take exercise.

In conclusion, I must acknowledge kind help from Dr. Ruedi of Davos, Dr. Holland of St. Moritz, and Dr. Wise of Maloja, in supplying information about the cases which had been entrusted to their charge.

*Postscript, July, 1888.*—In the discussion which followed it was urged that results equal to those of the high-altitude treatment of consumption had been obtained at lower levels by special attention to food, air supply, exercise, and medicine; and Dr. Dettweiler's statistics of cases of phthisis treated in his establishment at Falkenstein, in the Taunus, were instanced as examples. At my request Dr. Dettweiler kindly furnished me with a copy of his paper,<sup>1</sup> from which I extract the following facts: "Out of 1022 cases of phthisis, 132 quitted Falkenstein as "complete cures" and 110 as "relative cures," with still some symptoms and physical signs of disease. This yields about 13 per cent. "cures" and 10 per cent. "relative cures," and a total of 24·2 per cent. greatly improved. Dr. Dettweiler's cases, as far as they are given, do not appear to differ much from mine, and are certainly not more unfavorable, and the average length of time passed at the health resort is about the same in both sets of cases. But the high-altitude results yield 41·13 per cent. "cures" against Dr. Dettweiler's 13 per cent., and 82·25 per cent. improved (more or less) against Dr. Dettweiler's 24·2 per cent. Even among his "cures," the after-fate of only 98 could be ascertained, which throws some doubt on their right to be regarded as such; 72 were cases of arrest and remained so up to the present date, but 12 had relapsed and recovered, 3 had relapsed and not recovered, and 11 had died. It is needless to say that the high-altitude results need not fear comparison with such statistics.

<sup>1</sup> Bericht über zwei und siebenzig seit drei bis neun Jahren völlig geheilte Fälle von Lungenschwindsucht, 1886.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 382.)

ON THE  
VALUE OF THE TUBERCLE BACILLUS  
IN CLINICAL DIAGNOSIS.

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To some perhaps it may seem superfluous to argue in favour of the value of examining the sputum for tubercle bacilli in all cases of disease of the respiratory organs of uncertain nature. We believe, however, that in England the routine investigation of the sputum in doubtful cases is by no means generally practised. It is not denied that many observers in this country are as fully persuaded of the importance of examining the sputum as are the great bulk of Continental practitioners. But it must be admitted that a not inconsiderable proportion of English medical men still maintain an attitude of distrust towards this method of diagnosis. Considering the very great importance of this question it cannot be said that it has been made the subject of undue discussion. The clinical side of the subject has not been discussed at any of the

societies in London since 1883, when Dr. Whipham introduced the debate at the Medical Society. At that time Koch's discovery was quite recent, and sufficient time had not elapsed to allow a just estimate to be formed of the value of the sputum test in all its bearings. Since then our knowledge of the subject has been increased and consolidated, though some of the hopes at first expressed have not been realised. Important clinical contributions have been made by Drs. Whipham, Theodore Williams, Gabbett, Hunter Mackenzie, Dreschfeld, Heron, and others in this country, but the number of publications has not been large.

On the other hand, numerous Continental observers have written on this subject. Ehrlich, Balmer and Fraentzel, Leyden, Guttman, Ziehl, Gaffky, B. Fraenkel, Heitler, Rutimeyer, Frisch, May, Biedert, Germain Sée, Hugueny, D'Espine, and a host of others have brought forward the results of prolonged and laborious investigations. From their observations as well as from those of our own countrymen, it may be affirmed that Koch's bacilli can be detected in the sputum of all cases of phthisis at some period of the disease. Isolated instances have, it is true, been recorded where the bacilli could never be detected in spite of repeated examination, although the necropsy proved that the cases were undoubtedly tuberculous (two cases by Leyden and one by Ziehl).

Such cases are unquestionably worthy of consideration, though their number may be said to be almost insignificant. The statement which was made early in the history of the subject, that a direct proportion exists between the number of bacilli in the sputum and the severity and activity of the disease, is now for the most part admitted to be erroneous. The importance which was assigned to the morphological character of the bacilli, especially the appearance of spores, is no longer insisted upon.

The most disappointing result of all the labour that has been expended on these investigations is the generally

admitted failure of the sputum test for purposes of prognosis. We may well, however, be thankful if the diagnostic importance of the bacilli has been established upon a firm basis, which we believe to be the fact.

Some clinical authorities perhaps would still dispute this proposition altogether. The general truth of this conclusion, however, appears to be accepted by the immense majority of observers, and may be regarded as abundantly proved by the numerous and careful investigations that have been alluded to.

At the same time it is probably true that there are many physicians who recognise the value of this method of examination in a certain measure, but attach only a modified value to it. It is urged by such critics that the ordinary methods of physical investigation, combined with a careful consideration of clinical facts, are more trustworthy than the sputum test. Now, we have not the slightest intention of disputing the accuracy of this statement for the large majority of cases, where indeed, as it seems to us, the examination of the sputum plays quite an unimportant *rôle* in diagnosis. But the most experienced observers will be the first to admit that in certain instances, in spite of the most careful physical examination and clinical study, it may be impossible to arrive at a positive conclusion, or at the most the diagnosis may be a matter of probability.

If in such cases we find tubercle bacilli in the sputum we are justified in affirming the existence of tuberculosis; in other words we are enabled to convert an uncertainty, or a conjecture, into a positive assurance. The extreme importance of coming to a definite conclusion in special circumstances, without waiting for the progress of the case to decide, need hardly be insisted upon. A more important objection has been urged by Leyden, who points out that the appearance of the bacilli in the sputum *late* in the history of a case may lead us to misinterpret the nature of the affection, and to attach undue importance to what is only an accidental complication of the original



disease. This is perfectly true, and the warning is a valuable one. But a careful consideration of the history and progress of the disease, together with the physical condition of the patient, will prevent us from attributing too much significance to the detection of the tubercle bacilli in such cases. Illustrations of this fact will be given presently.

The object of the present communication is not to contribute a further proof of the general truth of the proposition which has been stated, but to bring before the Society the results of observations carried on during the last five years, as to the value of the sputum test in those cases alone in which the ordinary methods of diagnosis did not warrant a definite conclusion.

Striking cases have been described by various observers where the detection of tubercle bacilli made a positive diagnosis possible, in the complete absence of physical signs of pulmonary disease, or when the signs were anomalous, but such observations are only to be found scattered through the voluminous literature that has accumulated. It is believed that the facts now to be laid before the Society warrant a certain broad grouping of doubtful cases which has a practical value.

We must briefly allude to the methods we have adopted, though we make no claim to originality in this respect. The *sample* of sputum to be examined is of considerable importance. In every case where it was possible we obtained the *morning* sputum, as it was found that this gives the most trustworthy results. As others have remarked, the sputum expelled on waking in the morning represents the secretions that have accumulated for some hours in the larger air-tubes, and which are comparatively easily voided without hawking, which often results merely in the removal of pharyngeal mucus. A purely mucous sputum rarely yields tubercle bacilli, though exceptions are found to this rule. The most suitable portions for examination are the little opaque strings or fragments which are often to be seen in what at first sight appears

to be a purely mucous secretion. When the sputum is purulent or muco-purulent the thick whitish yellow pellets should be chosen. Where the sputum contains a large amount of saliva, or where it mainly consists of bronchial secretion, owing to concomitant bronchitis, it is very easy to arrive at a negative result erroneously. On the contrary, in such unfavorable samples of sputum as those just described, it is not unfrequently possible to detect bacilli by a careful selection of suitable portions. A convenient plan is to pour the sputum into a flat glass dish, and examine it against a dark background. Variations in the discharge of the bacilli are not uncommonly met with, and it is well in doubtful cases to examine the sputum at different periods of the day.

The most promising portion having been selected, a small piece is removed with a platinum wire bent into a small loop at one end, a needle, small scalpel, or fine-pointed pair of scissors, and spread in as thin a layer as possible on the surface of three or four cover-glasses. When the bacilli are likely to be few, or when they have not been previously detected, it is advisable to make preparations from different parts of the same specimen of sputum.

The cover-glasses are allowed to dry, and are then passed through the Bunsen flame in the usual manner, to coagulate the albumin, after which they are stained. After having made trial of various dyes, we find that the sharpest contrast is obtained by staining the bacilli red and the other constituents of the sputum blue, in preference to using a blue or violet stain for the bacilli, and a brown dye for the other elements.

The Weigert-Ehrlich (an aniline solution of fuchsine), or Ziehl's fluid (a carbolic acid solution of fuchsine) were employed for staining the bacilli, and a watery solution of methylene blue was the contrast dye.

In the case of the first solution of fuchsine the specimens were stained for twelve to fourteen hours as a rule, but when Ziehl's fluid was used the time allowed was

about one hour. In doubtful cases more prolonged staining was employed, and the preparations were heated gently. The ordinary nitric acid solution, 1 part in 3, was used for decolourising. No alcohol was allowed to touch the specimens, as the staining seemed to be sharper when the excess of the various dyes is removed by washing with water only. After the last washing the cover-glass is allowed to dry, and is then mounted in a drop of Canada balsam dissolved in benzol. A little practice enables one to secure good staining, which greatly facilitates the recognition of the bacilli, especially when their number is very small. For the examination of the preparations, it is absolutely necessary in doubtful cases to have an Abbé's or some similar condenser, a one twelfth oil immersion lens, and a very good light. It has frequently happened that specimens of sputum which have been passed by careful observers as containing no bacilli, have been referred to us for further examination, when bacilli have been detected, often in very small numbers. This we believe was mainly due to our invariable adoption of the three precautions just mentioned in all doubtful cases.

It has been asserted that the presence of elastic fibres in the sputum is of more value in diagnosis than the tubercle bacilli. This is entirely at variance with our experience. In many cases of advanced and progressive phthisis it is very difficult and often impossible to find elastic fibres, whereas we have hardly ever failed to find bacilli in similar circumstances, though their number may vary greatly. Apart from the greater facility with which the bacilli may be discovered as compared with elastic fibres, it must be remembered that the expectoration of elastic tissue merely shows that destruction of lung-structure is going on, without giving any information as to the nature of the disease. Tubercle bacilli, on the other hand, are only found when the process is tuberculous. Leyden and others have found that although bacilli may be detected without elastic tissue in cases of phthisis, the latter is never present without bacilli. This statement is

in accord with our experience. Before proceeding to describe the cases on which our remarks are based, it should be stated that the term "phthisis" is used in the sense of a tuberculous disease, *i. e.* an infective disease which tends to invade both lungs, and to give rise to specific lesions in other parts of the body.

The cases with which we are more especially concerned may be divided conveniently into five clinical groups:

- I. Cases with no physical signs of pulmonary disease.
- II. Cases of laryngeal disease of uncertain nature, without definite pulmonary signs.
- III. Cases with signs of bronchial catarrh, with or without emphysema.
- IV. Cases with physical signs of pleurisy.
- V. Cases with physical signs of doubtful import.
  - (a) Anomalous physical signs.
  - (b) Apex signs of uncertain nature.
  - (c) Signs confined to, or most marked at, the base.

A few cases illustrating each group will now be described in brief abstract.

The total number of cases on which the present communication is mainly based, numbering 100, have been tabulated as shortly as possible, and will be found at the end of the paper.

#### *I. Cases without Physical Signs. (Total 8.)*

CASE 1.—Mr. P—, æt. 30. Bronchitis a few months previously. Some cough and expectoration since then, with loss of appetite.

General condition good. Pharynx granular and congested. Chest repeatedly examined. No physical signs of disease whatever. The patient was not seen for about twelve months, when he again came under observation, suffering from the same symptoms. The chest was again

examined with a negative result, and the sputum contained no tubercle bacilli.

As he was going to the seaside, he brought another sample of sputum with him the day before he left town, and on this occasion special attention was directed to the chest, as he complained of a sharp pain in the side. No physical signs were detected, and he left London.

The next day the specimen of sputum was examined, and a few tubercle bacilli were detected.

Two months later, on his return from the seaside, there were subcrepitant râles at both apices, with weak breath-sounds, and the sputum contained a considerable number of bacilli. Physical signs of phthisis became more marked, and the diagnosis was confirmed by several distinguished physicians. The patient wintered at the Engadine without much profit, and on his return the following spring gradually began to fail, and died of confirmed phthisis at the end of last year, eighteen months after tubercle bacilli were first found in the sputum.

CASE 2.—Emily M—, æt. 25. Rheumatic fever ten years ago. Ailing fifteen months since last confinement, with weakness, shortness of breath, slight cough, and expectoration. Five weeks ago she noticed some lumps in her neck, and two weeks later in the right armpit also. The patient was anæmic and very weak. Temp. 102°. Clusters of enlarged glands in the right side of the neck and in right axilla. No glandular enlargement elsewhere. Abdominal organs not enlarged.

No signs of disease of the lungs or heart.

Sputum scanty, and contained tubercle bacilli in small numbers. Two days later the bacilli were found in large numbers.

No physical signs could be detected till a week later, when the breath-sounds were found to be slightly weaker at the right than the left apex, and a small patch of con-

solidation was discovered in the right interscapular space. These signs gradually cleared up, but a fortnight subsequently some muffled subcrepitant râles were heard at the right supraspinous fossa.

No further change in physical signs has yet taken place. The temperature is irregular, ranging from  $102^{\circ}$  to  $99^{\circ}$ . The appetite and general strength have improved. Patient remains under observation.

CASE 3.—Sarah B—, æt. 27. Winter cough for two years. Ailing for four weeks with cough, slight expectoration, dyspnœa, and wasting. Hæmoptysis on three occasions, not profuse, the first attack four months ago. Nutrition fair. No physical signs.

Bacilli in sputum in small numbers. Sputum almost entirely mucous. A fortnight later bacilli still more scanty. Still no physical signs.

At a third examination a few weeks later no bacilli could be found. Since then the sputum has become so scanty that it is almost impossible for the patient to bring a sample for examination. No marked change has occurred in the general condition, but the resonance at the right supraspinous fossa is now somewhat high pitched, and scanty subcrepitant râles are audible in this region. Patient remains under observation.

## II. *Cases of Laryngeal Disease of Uncertain Nature, without Definite Pulmonary Signs.*

CASE 4.—Jesse C—, æt. 51. Previous history good. Six months ago a slight cough developed, and his voice became weak. No emaciation and no dyspnœa. No physical signs in chest.

Larynx: General reddening. Both vocal cords show an irregular, ulcerated surface. No tubercle bacilli found in sputum or in secretion taken from larynx on several occasions. The ulceration gradually healed, but small

tumours developed in the region of the anterior commissure of the cords. Portions of the growth were removed with laryngeal forceps, but on microscopic examination they were found to consist simply of finely granular necrotic material.

After removal a distinct but small subglottic tumour could be seen below the right cord. A whitish mass was subsequently seen on the upper surface of the left cord. A few months later the temperature rose slightly, the patient complained of pain in the right side, and some crackling râles were heard at the right base in front. Tubercle bacilli were now found in the sputum for the first time. Tracheotomy was performed next day owing to increasing obstruction of the larynx, and the granulations were scraped away as far as possible. Microscopical examination of pieces removed showed merely a finely granular necrotic structure. Tubercle bacilli could now always be detected in the sputum. At a second operation, which became necessary some months later, the growth proved to be tubercular on microscopic examination.

Death twelve months after first symptoms.

*Post-mortem.*—A small cavity at the apex of the right lung, scattered tubercular nodules below. Recent consolidation and excavation of left apex. Diffuse tuberculous ulceration of the larynx.

CASE 5.—James S—, æt. 60. Ailing six months, with cough and dysphagia. Inspiratory stridor and dyspnoea. Tracheal breathing over the manubrium, faintly heard also at the apex of each lung. Breath-sounds weak.

Larynx: Much swelling of aryepiglottic folds. A small pinkish rounded growth on the internal surface of the left arytenoid cartilage. Vocal cords fixed in the median position, being separated by not more than one sixteenth of an inch.

*Diagnosis.*—Laryngeal new growth. Infected tracheal glands. Tubercle bacilli were found in the sputum.

Dyspnoea became so urgent as to necessitate tracheotomy. Death five weeks after the operation.

*Post-mortem.*—Old fibro-caseous patches at the apices of both lungs, and a few small cavities. Miliary tuberculosis of the lower parts. Extensive tubercular infiltration and ulceration of the larynx. A small tubercular tumour springing from the left arytenoid region.

CASE 6.—Thomas D—, æt. 39, hawker, a fairly well-nourished and muscular man. Ailing four months with cough, slight expectoration, hoarseness, and loss of flesh. Inspiratory stridor and dyspnœa. Tracheal breathing over the sternum and weak tubular breathing at both apices. No distinct impairment of resonance. Obscure râles were heard once or twice, now at one now at the other apex.

Larynx: Slight swelling over the arytenoid cartilages, and some irregularity of the interarytenoid fold, from which a small tag-like process projects. Vocal cords fixed in the median position one eighth of an inch apart. Both cords somewhat swollen and granular, their posterior parts coarsely nodulated. No ulceration.

Tubercle bacilli were found in the sputum at four successive examinations. Tracheotomy was performed on account of increasing stenosis of the glottis. Ten days later distinct subcrepitant râles were heard at both supraspinous fossæ. The patient remains under observation.

### III. *Signs of Bronchitis with or without Emphysema.* (Total 15.)

CASE 7. *Signs of Bronchitis; hæmoptysis; bacilli detected in the blood expectorated.*—Lewis D—, æt. 25, previous health good. Four weeks ago caught cold and cough developed, but he was not laid up at first. Eighteen days later profuse hæmoptysis occurred, and two more attacks of hæmorrhage followed within the next twenty-four hours; over two pints of blood were said to have been lost. Owing to the condition of the



patient no thorough examination was possible, bubbling râles being heard all over both lungs. Tubercle bacilli were found in the blood which the patient brought up.

On admission to the hospital a few days later, weak breath-sounds with prolonged expiration and occasional crepitation were heard at the left apex.

Physical signs developed rapidly on both sides ; bacilli were found in increasing numbers in the sputum, and death occurred twelve weeks after the first hæmoptysis.

*Post-mortem.*—Scattered caseous nodules in the right lung, extensive recent excavation in the upper part of the left lung and caseous nodules.

CASE 8. *Signs of emphysema and bronchitis.*—Clara Q—, æt. 25. Cough for six months, slight hæmoptysis. Chest hyper-resonant everywhere, bubbling râles diffused over both lungs. Tubercle bacilli in the sputum in large numbers.

The bronchitis gradually cleared up, but signs of excavation developed at the right apex.

CASE 9. *Signs of bronchitis.*—George Y—, æt. 42. Winter cough off and on for ten years, continued cough the last twelve months with expectoration and some loss of flesh. Alcoholic tendencies.

The patient was a short, stout, muscular man, with a very fine deep chest. Scattered sibilant sounds and muffled bubbling râles all over the right lung, and at the left base. Pharynx and larynx deeply congested. Sputum muco-purulent, containing a few tubercle bacilli. Three months later there was slight dulness at the right extreme apex with weak breath-sounds, prolongation of expiratory sound, and scanty subcrepitant râles. No change in symptoms ; patient thinks he has lost flesh. Still under observation.

IV. *Physical Signs of Pleurisy.* (Total 12.)

CASE 10.—George H—, æt. 32. Cough for nine months with slight expectoration and some loss of flesh. Patient very anæmic and looks ill. On the right side dulness from the spine of the scapula to the base, extending laterally to the mid-axillary line. Resonance slightly impaired above the clavicle. An exploratory puncture at the base yielded clear serous fluid. Numerous tubercle bacilli were found in the sputum on several occasions. The fluid became absorbed, but marked signs of phthisis developed in both lungs. The patient was treated by massage and made considerable improvement.

CASE 11.—John E—, æt. 17. “Cold and cough” for six months and pain in the right side. Signs of pleural effusion on the left side reaching up to the third rib. Tubercle bacilli detected in the sputum frequently but in small numbers. The effusion became absorbed and improvement followed. Slight dulness and weak breath-sounds at the left base were the only abnormal signs when the patient left the hospital. Four months after his discharge the patient wrote to say he was doing well and was about to go to work.

CASE 12.—Henrietta B—, æt. 36. Cough for three years, worse the last three months and accompanied with pain in the right side. Marked signs of effusion into the right pleura. Three and a half pints of serous fluid were removed by paracentesis. Empyema developed and a free incision was made. The pus contained numerous tubercle bacilli. Death.

*Post-mortem.*—Empyema on the right side, lung collapsed and containing scanty, softening, fibro-caseous nodules. One nodule immediately beneath the pleura had discharged its contents into the pleural cavity, without any communication between the lung and pleura having

occurred. Similar nodules and recent miliary tuberculosis in the left lung.

CASE 13.—John M—, æt. 34. Thirteen months ago cough began and was soon followed by hæmoptysis. Physical signs of pleuritic effusion existed on the right side, a few indistinct râles being heard at the apex. Free incision. The purulent fluid removed contained tubercle bacilli. The bacilli could not be detected in the sputum at first, but were afterwards found several times both in the sputum and in the purulent discharge. The wound never healed, and a subsequent operation failed to improve matters. The patient is still alive, three years after the first operation, but his health is failing, and marked signs of phthisis have been present in both lungs for some months.

#### V. *Physical Signs of uncertain import.* (Total 56.)

##### (a) Anomalous or indefinite signs. (18.)

CASE 14. *Signs simulating a localised pneumothorax.*—Samuel F—, æt. 24. Shortness of breath and wasting for eighteen months. Laid up with “inflammation of the lungs” eight months ago for thirteen weeks. Slight flattening in left subclavian region with hyper-resonance and weak breath-sounds over the whole upper lobe. No other physical signs. Tubercle bacilli in the sputum in fair numbers. The hyper-resonance became more marked over the left upper lobe with almost complete abolition of breath-sounds. No bell-sound to be heard. Breathing less weak at the base. Scanty crackling sounds in the left axilla and rhonchus on the right side.

? Localised pneumothorax, or, a large vomica with obstructed bronchus. Diagnosis quite uncertain.

The sputum, which was scanty, was examined four times but only once contained bacilli.

The patient complained mainly of shortness of breath,

but was not confined to his bed. No change in physical signs occurred and he was lost sight of.

CASE 15. *Signs simulating aneurism.*—John L—, æt. 34. No history of syphilis or rheumatism. Fourteen months ago he caught a chill, which was followed by cough, pain in the right shoulder, shortness of breath, and repeated but slight attacks of hæmoptysis. Slight dulness in the first right intercostal space close to the sternum, and to a less extent in the supraspinous fossa. Over this area prolonged expiration, increased vocal resonance, and scanty subcrepitant râles. Distinct pulsation close to the sternum, but no tumour. A short systolic murmur was present in the second space, and the aortic second sound was very loud. Tracheal breathing was heard over the manubrium sterni. The only other abnormal physical signs were dulness and weak breath-sounds in the right axilla over the middle lobe.

The sputum was examined three times for tubercle bacilli and twice for elastic tissue with a negative result. At a fourth examination bacilli were found in fair numbers.

The patient improved somewhat and remains under observation.

The dulness now extends rather further outwards under the right clavicle, and tracheal breathing is now only faintly heard over the sternum. Otherwise the physical signs remain unaltered.

CASE 16. *Mammary carcinoma, (?) intrathoracic growth.*—Harriet M—, æt. 53. Tumour in the right breast for three years. Gradual wasting and cough the last ten months. Carcinoma of right breast well marked. Signs of general pleuritic thickening over the right lung, and doubtful cavernous signs at the lower aspect of the upper lobe in front. Tubercle bacilli in the sputum in very small numbers on two occasions.

The patient was under observation for many months without any change occurring in the physical signs. The

mammary disease made little or no progress. During her stay in the hospital acute glaucoma developed for which iridectomy was performed, and not long afterwards a strangulated femoral hernia was cured by operation. Nevertheless, when the patient finally left the hospital her condition was better than when she was admitted.

CASE 17. *Aneurism of the aorta ; secondary tuberculosis.*—Richard A—, æt. 36. Gonorrhœa and stricture twenty years ago. Gradually increasing shortness of breath the last twelve months. Face congested. Slight inspiratory stridor. Tracheal breathing over manubrium, with a short systolic murmur and a loud second sound. Breath-sounds weaker on right than left side. Larynx and trachea congested. No obstruction visible. Albuminuria.

*Diagnosis.*—Aneurism of aorta. Stenosis of trachea and right bronchus.

The dyspnœa gradually increased and assumed a paroxysmal character. After some months the question of a “new growth” was raised. Slight impairment of resonance was noted throughout the whole right side without further change in physical signs.

During the last few weeks of his life the patient expectorated with great difficulty a little tenacious brownish-red sputum. The sputum was now examined and found to contain a large number of tubercle bacilli. The existence of pulmonary tuberculosis, which was thus proved, was regarded as a secondary complication not affecting the original diagnosis. Death occurred twelve months after the first examination from failure of the heart following an agonising attack of dyspnœa.

*Post-mortem.*—A medium-sized, thin-walled aneurism of the aortic arch, compressing the trachea and both bronchi, especially the right. General pleuritic thickening and adhesion over the right lung, with a few small recent cavities in the apex, and tuberculous disease limited to the upper lobe. Granular kidneys.

V. (b) *Apex Signs of uncertain nature.* (Total 22.)

CASE 18. *Ulcerative endocarditis, (?) phthisis.*—Harriet P—, æt. 22. No history of rheumatism. Ailing seven months with cough, expectoration, dyspnoea, and wasting. Systolic murmur in aortic and mitral regions, and a double murmur over pulmonary area. Resonance high pitched at apices of lungs with puerile breathing. Moist crepitation at bases. Bacilli in the sputum.

Some weeks later crepitation became localised at the right supraspinous region. Subsequently signs of excavation appeared at both apices, and the case became further complicated by the development of dry pleurisy, pericarditis, and albuminuria. Irregular pyrexia throughout. Death four months after admission.

*Post-mortem.*—Ulcerative endocarditis of both sides of the heart, a very small old cavity at the right apex, and a few thin-walled cavities as big as peas, with some small scattered caseous nodules at the left apex.

CASE 19. *Cirrhosis and bronchiectasis ; secondary tuberculosis.*—Henry F—, æt. 33, lately discharged from the army. Caught cold in Egypt ten months ago from sleeping in a wet bed. Since then cough, profuse hæmoptysis on several occasions, and wasting. Expectoration, offensive almost from the first, coming up in large quantities when he lay on the left side. Signs of consolidation and excavation on the right side from the apex to the fourth rib in front, and at both supraspinous and infraspinous fossæ posteriorly. Bronchial râles under left clavicle. The fœtor of breath was so extreme that the patient had to be removed to a ward by himself. Sputum very profuse and extraordinarily foul. The question was raised as to the advisability of attempting to drain the large cavity by surgical means, but the sputum was found to contain tubercle bacilli, and all idea of an operation was abandoned.

Diagnosis bronchiectasis, phthisis secondary. Death from exhaustion after some weeks of irregular pyrexia.

*Post-mortem.*—Localised empyema at right base. Cirrhosis and excavation (? bronchiectatic) of the right lung with general bronchial dilatation and some scattered caseous nodules. Scattered caseous nodules in left lung, but no other disease. Tuberculosis appeared to be a secondary complication.

CASE 20. *Enlarged glands simulating lymphadenoma at first. Slight apex signs.*—George B—, æt. 41, cough off and on for four years since an attack of typhoid fever. Greatly enlarged glands on the neck, raising the question of lymphadenoma for a time. Patient was a stout muscular man.

Very slight impairment of resonance at both apices with weak breath-sounds and doubtful râles at the right apex, which soon disappeared entirely, the other signs remaining unchanged. Sputum examined three or four times with a negative result; at last a few bacilli were detected. The patient disappeared for eighteen months, but on re-examination no change had occurred in the physical signs (dulness and weak breathing at each apex without râles). The sputum now contained bacilli in fair numbers.

Eight months later the patient had lost ground somewhat and scanty subcrepitant râles were heard at both apices. Still under observation. Cervical glands much smaller.

V. (c) *Physical Signs confined to, or most marked at, the base.* (Total 16.)

CASE 21. *Primary tuberculosis of the base.*—John W—, æt. 50. Five months ago caught cold, had shivering fits, and was confined to bed for thirteen weeks with pains in the back and side, cough, expectoration, and loss of flesh.

On admission, respiration was very rapid, and temperature  $103^{\circ}$ . Lungs generally hyper-resonant with the exception of the posterior bases, where there was slight dulness. At the right base weak breath-sounds with prolonged expiration and diminished vocal fremitus. At the left base tubular breathing and increased vocal resonance. Subcrepitant râles at both apices.

Tubercle bacilli in the sputum. At first their number was not great, but subsequently they were found in abundance.

The patient steadily went down hill, signs of phthisis developed in the upper parts of the lungs, and cavernous signs were detected at both bases.

*Post-mortem.*—True tuberculous phthisis, originating at the bases of the lungs.

CASE 22.—Oliver D—, æt. 18. Cough for eighteen months, onset gradual. Hæmoptysis soon after cough developed. Expectoration rather profuse and paroxysmal. No foetor.

The patient was a rather thin, pale lad, but was in fairly good health, and had not lost flesh.

On examination, there was dulness on the right side from the fifth rib downwards, most marked at the base, where there were signs of excavation. No other abnormal signs on either side.

*Diagnosis.*—Cirrhosis of the lung or basic phthisis. The sputum contained tubercle bacilli in large numbers. It was subsequently ascertained that the patient had been in the hospital a few months previously, when the diagnosis had been "chronic pneumonia."

The patient has improved slightly and remains under observation.

CASE 23. *Cirrhosis and bronchiectasis; secondary tuberculosis.*—Sydney B—, æt. 10. Cough six years, since an attack of measles. Breath offensive the last three years. Expectoration very foul, and brought up periodically in large "gushes."



Fœtor of breath extreme, odour faintly fæcal. Fingers clubbed. Slight dulness all over right back, most marked at base; coarse bubbling râles over the whole right lung, with weak breath-sounds. At the base the chest wall is somewhat retracted, and vocal fremitus and resonance are diminished. A few tubercle bacilli in the sputum.

*Diagnosis.*—Cirrhosis of the lung and bronchiectasis. Secondary tuberculosis. Signs of a cavity developed at the right apex six weeks later, and the general condition gradually deteriorated. The patient left the hospital and died at his home some months later. No post-mortem.

Before proceeding to discuss the conclusions which the above facts suggest, it may be stated that the 100 cases that are narrated represent only a portion of the total number that have been examined. Many other cases might be quoted where doubts as to diagnosis were removed by the discovery of bacilli in the sputum. We have endeavoured to restrict our observations to cases where the diagnosis presented real difficulties. We freely admit that many of our cases were strongly suggestive of phthisis. But it is one thing to suspect the existence of this disease and another to be able to say that it is actually present. Herein lies the great value of examining the sputum.

No negative cases have been included, except when the conclusions have been tested by post-mortem examination. The importance of positive evidence and the comparative worthlessness of negative results in this department of diagnosis can hardly be too much insisted upon. At the same time a negative result after repeated examinations, possesses a certain confirmatory value if it is in accord with the general clinical features of the case. It seems impossible to doubt the significance attaching to the discovery of the tubercle bacillus from a diagnostic point of view. Such facts as those that have been narrated demonstrate the possibility of correctly diagnos-

ticating many cases where the existence of tuberculosis is masked by certain well-recognised conditions.

Whatever views may be held as to the pathogenic properties of Koch's bacilli, their presence in the expectoration of cases of phthisis and their absence in all other diseases seems hardly to be questioned by anyone now.

It has indeed been stated by G. Zahn that tubercle bacilli were found by him in the sputum of cases of bronchitis in which post-mortem examination demonstrated the absence of tuberculosis. Further details of his cases are wanting, as it has not been possible to obtain his original paper.

Leyden and Ziel suggest that Koch's bacilli may occasionally be found in the sputum of cases of simple bronchitis, especially in hospitals. That is to say, the bacilli may be detected in the bronchial secretions soon after they are inhaled, and before they are destroyed by the action of the living tissues. This of course presupposes that the bacillus is ubiquitous. We have never been able to verify this statement, although numerous cases of bronchiectasis with profuse secretion have been specially examined in this direction, the patients having in many instances been for months in the hospital. In no case where post-mortem examination was made was tuberculosis proved to be absent when the sputum during life had contained Koch's bacilli.

No constant relation was found to subsist between the number of bacilli expectorated and the severity of the disease. As a rule, bacilli are numerous in acutely progressive cases, but exceptions are not rare. Bacilli are sometimes very scanty when pyrexia is high, whereas in very chronic disease their number may be very large. No definite importance can be attributed to the presence of groups of bacilli; nor can we confirm the statement of Dr. Hunter Mackenzie that the bacilli are always numerous when the tubercular ulceration of the larynx is present. In several well-marked instances of this type bacilli were decidedly scanty. Whether it is necessary

to assume the existence of a softening process in the lung in all cases where bacilli are expectorated, we are not prepared to say. But in some cases of acute miliary tuberculosis without softening, microscopical examination has revealed minute bacillary ulcers in the bronchioles, which probably discharged some of their bacilli with the expectoration during life. In these cases we had no opportunity of clinical examination. We may perhaps be allowed to repeat that we have no wish to exaggerate the importance of the sputum test, as compared with the method of auscultation and percussion.

Physical examination of the chest is infinitely the more valuable means of diagnosis, but we maintain that cases are not uncommonly met with where this method is found wanting, and may be most usefully supplemented by the investigation of the sputum. This proposition seems to be justified by the facts that have been described.

The difficulty presented by cases where there is a complete absence of physical signs in spite of definite pulmonary and constitutional symptoms, is too well known to need further comment.

The laryngoscopic diagnosis of tuberculous from syphilitic disease of the larynx is at times no easy matter, especially where the physical signs of pulmonary disease are absent or equivocal, and where a well-marked history of previous syphilis is forthcoming. A similar difficulty may arise in the diagnosis of laryngeal tuberculosis from carcinoma. This fact is well illustrated by a case narrated in the 'Lancet' of 1887, where the absence of physical signs, and the appearance of the larynx, seemed to point to epithelioma, and extirpation of the larynx was practised, the patient succumbing a few days afterwards.

In this instance an examination of the sputum would probably have induced the surgeon to prefer tracheotomy to so serious an operation as excision of the larynx.

The uncertain value of auscultation and percussion in severe obstructive laryngeal affections of any description cannot be too strongly insisted upon. In all such cases

the examination of the sputum has an important bearing not only on diagnosis but indirectly on treatment and prognosis also.

We may briefly allude in the case of pleuritic effusions to the fallacies involved in auscultation of the uncompressed portions of the lung or of the opposite lung, to the uncertain value of pyrexia, and to the fact that complete absorption of the fluid is not incompatible with the presence of pulmonary tuberculosis as illustrating the desirability of some further test.

The fact that phthisis may be completely masked by bronchitis and emphysema should in all suspicious cases of this kind direct attention to the sputum, which will often explain much that would otherwise be obscure.

The last class, to which we would especially direct attention, comprises the large group of cases in which the base of the lung is exclusively or mainly concerned. The diagnosis of simple cirrhosis and bronchiectasis from tuberculous disease is the difficulty that most commonly occurs, but pulmonary abscess, empyema, hydatid disease of the lung or liver, abscess of the liver, mediastinal growths, and other conditions, have all to be considered in individual cases.

The well-known rarity of primary tuberculosis of the base, and the almost invariable presence of apical signs also, will usually, but not always, prevent mistakes being made. Here again the bacillary test is of the utmost importance.

Finally, we would venture once more to emphasize the necessity for great care in the application of this test. Unless the sputum be examined with the precautions that have been alluded to in all doubtful cases, it is better not to examine it at all, lest failure to detect the bacilli should prejudice the conclusions arrived at on other grounds. It may be necessary in special instances to examine the sputum ten or twelve times before a positive result is obtained, but this is the exception. We have only to

add that the bacilli have been found to be of little service for purposes of prognosis.

Complete disappearance of the bacilli from the sputum would of course be a most important element in prognosis if combined with marked improvement of the general condition and physical signs. Unfortunately this concurrence of events is extremely rare. On the other hand, a diminution or increase of the number of bacilli in the sputum possesses little importance when taken alone. For it must be remembered that the number of these micro-organisms found in the sputum is not necessarily a measure of the activity or extent of the disease, but is mainly a question of discharge.

Practically, the prognosis turns on the general condition of the patient more than on anything else, and although the examination of the sputum may corroborate the verdict given on general grounds, it is not of itself of much value.

### *Summary of Cases.*

*Abbreviations.*—D. or d. = dulness; crep. = crepitation; subcrep. = subcrepitant râles; S. S. F. = supra-spinous fossa; T. B. = tubercle bacilli.

The mark \* indicates that the case is described more fully in the previous part of the paper.

No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
	GROUP I.					
1	*Mr. P.	30	Nil	—	T. B.	Marked signs of phthisis developed. Death.
2	Arthur R.	18	Nil	—	T. B.	Recovery.
3	Mr. A.	30	Nil	—	T. B.	Recovery.
4	Jas. B.	30	Nil	—	T. B.	Recovery.
5	Edward R.	36	Nil	—	T. B.	Dulness appeared at right apex. Patient lost sight of.
6	John T.	21	Nil	—	T. B.	Unknown.
7	*Sarah B.	27	Nil	—	T. B.	Signs of phthisis developed.
8	*Emily M.	25	Nil	—	T. B.	Signs of phthisis developed.

No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
<b>GROUP II.</b>						
9	Annie H.	38	Nil	Ulceration, (?) syphilitic	T. B.	Pleuritic symptoms appeared; subcrep. right mammary region. Patient lost sight of.
10	John B.	42	Infiltration of right apex	Ulceration of larynx and tongue, (?) syphilitic	T. B.	Signs of phthisis appeared.
11	*Jesse C.	51	Subcrep. right anterior base	Ulceration and polypoid granulations, (?) syphilitic	T. B.	Signs of phthisis. Death. P.M.—Pulmonary and laryngeal tuberculosis.
12	*Jas. S.	60	Tracheal breathing over sternum	Infiltration, small tumour, fixation of cords, (?) new growth	T. B.	Signs of phthisis. Death. P.M.—Pulmonary and laryngeal tuberculosis.
13	*Thos. D.	39	Tracheal breathing over sternum	Ulceration, infiltration, and fixation of cords	T. B.	Signs of phthisis.
14	Geo. S.	40	Obscure crep. at apices	Ulceration, subglottic tumour, (?) syphilitic	T. B.	Signs of phthisis. Death. P.M.—Pulmonary and laryngeal tuberculosis.
15	Wm. E.	45	Tracheal breathing over sternum	Intense congestion and infiltration, (?) syphilitic	T. B.	Signs of phthisis. Death. No P.M.
16	Mary D.	26	Variable râles at apices	Ulceration, polypoid granulations (?) syphilitic	T. B.	Signs of phthisis.
17	Geo. S.	26	Limited dry pleurisy, left side	Slight ulceration, polypoid granulations, (?) syphilitic	T. B.	Crep. developed at right apex. Patient lost sight of.
<b>GROUP III.</b>						
18	Jas. B.	43	General rhonchus and sibilus	—	T. B.	Progressive failure of health. Lost sight of.

No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
19	Thos. S.	37	Scattered rhonchus; obscure râles right S. S. F.	—	T. B.	Signs of phthisis.
20	Harry W.	32	Hyper-resonance; diffused bubbling râles; slight d. left apex	—	T. B.	Lost sight of.
21	Jas. S.	62	Hyper-resonance; diffused bubbling râles left side	—	T. B.	Lost sight of.
22	Thos. H.	20	Hyper-resonance; scattered rhonchus	—	T. B.	Signs of phthisis.
23	Chas. H.	16	Hyper-resonance; slight d. right apex	—	T. B.	Signs of phthisis; very chronic course
24	John C.	25	Hyper-resonance; weak breath sounds; diffused bubbling râles	—	T. B.	Lost sight of.
25	Annetta G.	34	Hyper-resonance; scattered rhonchus	—	T. B.	Improvement; signs of emphy- sema persisted.
26	*Geo. Y.	42	Scattered sibilus and bubbling râles	Con- gestion	T. B.	Signs of phthisis at right apex
27	*Lewis D.	25	Diffused bubbling râles; patient recovering from an attack of hæmoptysis	—	T. B. in blood expecto- rated	Signs of phthisis. Death. P.M.— Pulmonary tuberculosis.
28	*Clara Q.	25	Hyper-resonance; diffused bubbling râles	—	T. B.	Signs of phthisis.
29	Henry C.	48	Hyper-resonance; diffused bubbling râles; some fine crep. left apex	—	T. B.	Lost sight of.
30	Mary H.	40	Hyper-resonance; diffused bubbling râles and rhonchi	—	T. B.	Signs of phthisis.
31	Samuel K.	53	Hyper-resonance; slight d. above right clav.; a few râles at apices	—	T. B.	Signs of phthisis.
32	Mr. H.	47	Scattered sibilus; very slight d. left apex	—	T. B.	Signs of phthisis.
GROUP IV.						
33	*Geo. H.	32	Right pleuritic effusion; paracentesis; serous fluid	—	T. B.	Signs of phthisis.
34	*John E.	17	Left pleuritic effusion	—	T. B.	Absorption of fluid. Recovery.
35	*Henrietta B.	36	Right pleuritic effusion; paracentesis; serous fluid; empyema developed	—	T. B. in pus from pleura	Death. P.M.— Tuberculosis of lung and pleura.

No.	Name.	Age	Physical signs.	Larynx.	Sputum.	Result.
36	*John M.	34	Right pleuritic effusion; empyema; free incision	—	T. B. in pus from pleura, and in sputum	Signs of phthisis.
37	Jas. D.	20	Thickening of pleura at right base	—	T. B.	Signs of phthisis.
38	Richard C.	43	Thickening of pleura at right base	—	T. B.	Signs of phthisis.
39	Elizabeth D.	44	Pleuritic effusion right side; dry pleurisy left base	—	T. B.	Lost sight of.
40	Wm. F.	52	Thickening of pleura at right base	—	T. B.	Signs of phthisis.
41	Alfred P.	20	Dry pleurisy and localised consolidation of middle third of left lung	—	T. B.	Signs of phthisis.
42	Samuel B.	43	Thickening of pleura at both bases	—	T. B.	Signs of phthisis. Death. P.M.—Pulmonary tuberculosis.
43	Abel P.	49	Thickening of pleura at left base; slight d. and subcrep. right apex	—	T. B.	Signs of phthisis.
44	Robert S.	44	Right pleuritic effusion; empyema; incision	—	No T. B.	Epileptiform convulsions, (?) due to cerebral abscess. Death. P.M.—Lymphosarcoma of mediastinal glands; secondary growth in brain; no tuberculosis.
GROUP V (a)						
45	*Samuel F.	24	Hyper-resonance and extreme weakness of breath-sounds over left upper lobe; (?) localised pneumothorax	—	T. B.	Physical signs persisted. Patient lost sight of.
46	Agnes P.	30	Stridor; tracheal breathing over sternum and apices; (?) stenosis of trachea	—	T. B.	Signs of phthisis. Tracheal breathing over sternum persisted.
47	Jesse T.	33	Slight d. left apex; disappearance of d. and development of hyper-resonance	Congestion	T. B.	No improvement. Patient lost sight of.
48	Thos. L.	29	Circumscribed consolidation in right axilla	—	T. B.	Improvement. Patient lost sight of.



No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
49	Chas. L.	42	<i>Signs simulating aneurism:</i> Weak breath-sounds on left side; obscure râles at apices; aortic second sound loud; pulse and pupils unequal	—	T. B.	Signs of phthisis and laryngeal tuberculosis developed.
50	*John L.	34	<i>Signs simulating aneurism:</i> D., pulsation, tubular breathing, and a loud aortic second sound to right of manubrium	—	T. B.	Signs of phthisis.
51	Walter S.	55	<i>Signs simulating aneurism:</i> Slight d., tubular breathing, pectoriloquy, systolic murmur, and loud aortic second sound to right of manubrium	—	T. B.	No change. Still under observation.
52	Henry D.	47	<i>Signs simulating aneurism:</i> Tracheal breathing over manubrium and apices; aortic second sound loud; pupils unequal	Congestion; no paralysis	T. B.	Signs of phthisis.
53	Arthur G.	28	Signs of consolidation in right interscapular space in course of descending bronchus	—	T. B.	Arrest. Recovery of health. Physical signs unchanged.
54	Richard D.	30	<i>Fugitive signs:</i> At first transient d. at apices, followed by temporary d. at right base; finally, d. at left base with subcrep. at both bases	—	T. B.	Failure of health. Lost sight of.
55	*Harriet M.	53	General thickening of right pleura; doubtful cavernous signs over lower part of upper lobe; old carcinoma of breast	—	T. B.	Slight improvement. Signs persistent.
56	Lewis M.	36	Localised bulging over right scapula with absolute dulness and bronchial breathing over same area; exploratory puncture with negative result (?) new growth	—	T. B.	Temporary improvement. Death at patient's home in country. No P.M.

No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
57	Albert S.	30	Circumscribed crackling sound about right nipple.	—	T. B.	Lost sight of.
58	Ellen C.	35	Occasional crackle in right axilla; resonance at right apex high pitched.	—	T. B.	Signs of phthisis.
59	Chas. K.	57	D. and cavernous signs limited to right interscapular space.	—	T. B.	Signs persisted. Lost sight of.
60	Geo. S.	45	Marked d. all over right side, breath-sounds weak, faintly tubular above with pectoriloquy	—	T. B.	Lost sight of.
61	Henry P.	20	Contraction and d. on left side, with cavernous signs throughout	—	T. B.	Lost sight of.
62	*Richard A.	36	Stenosis of trachea and right bronchus; systolic murmur and loud second sound at manubrium.	Con- gestion	T. B.	Progressive dyspnoea. Death. P.M.—Aneurism of aorta. Recent tuberculosis of lung.
(b)						
63	Elizth. R.	33	Consolidation of both apices; mitral stenosis	—	T. B.	Signs persist. Still under observation.
64	Ernest A.	20	Slight signs of consolidation at right apex; mitral stenosis	—	T. B.	Signs of phthisis became well marked.
65	Frank D.	26	Consolidation of right apex; mitral stenosis and incompetence	—	T. B.	Signs persistent.
66	Thos. E.	34	Consolidation of left apex; aortic incompetence	—	T. B.	Signs of phthisis became well marked.
67	*Harriet P.	22	Resonance high pitched at apices with puerile breathing; crep. at bases; aortic and mitral systolic murmur; double pulmonary murmurs	—	T. B.	Death. P.M.—Ulcerative endocarditis of both sides of heart. Pulmonary tuberculosis.
68	Charlotte B.	24	Consolidation of left apex; aortic incompetence	—	T. B.	Signs persistent. Slight improvement.
69	John B.	50	Slight d. right apex and subcrep. Hyper-resonance and sibilus on left side	—	T. B.	Signs of phthisis became well marked.

No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
70	*Henry F.	33	Consolidation and excavation of upper half of right lung. Diagnosis: bronchiectasis; phthisis secondary	—	Very foul. T. B.	Death. P.M.—Cirrhosis and bronchiectasis with excavation; secondary tuberculosis.
71	Wm. T.	35	Harsh inspiratory and prolonged expiratory sound at left apex, with subcrep.	—	T. B.	Lost sight of.
72	Robert W.	39	Slight d. right S. S. F.; breath-sounds weak; expiration prolonged, with subcrep. râles	—	T. B.	Lost sight of.
73	Wm. A.	25	Slight d. right apex; prolonged expiration and subcrep. at interscapular region	—	T. B.	Signs of phthisis became marked
74	Martha A.	32	Slight d. right apex, with prolonged expiration and subcrep.	—	T. B.	Lost sight of.
75	*Geo. B.	41	Very slight d. at apices, with weak breathing; greatly enlarged glands, suggesting "lymph-adenoma"	—	T. B.	Signs of phthisis developed; chronic course
76	Rebecca S.	26	Slight d. at apices with prolonged expiration and subcrep.	Congestion	T. B.	Lost sight of.
77	Wm. W.	40	Very slight d. left apex and base, with weak breath-sounds	—	T. B.	Lost sight of.
78	Wm. S.	44	Very slight d. over right upper lobe, with tubular breathing and pectoriloquy; no râles	—	T. B.	Lost sight of.
79	David P.	27	Very slight d. and weak breathing at left apex	Congestion and infiltration	T. B.	Signs of phthisis became well marked
80	Edward P.	27	D., weak tubular breathing, pectoriloquy, and subcrep. over left upper lobe	—	T. B.	Two attacks of hæmoptysis and gradual failure; signs unchanged
81	Benjamin B.	24	Slight d. right apex, followed by hyper-resonance, with blowing breathing and occasional râles	—	T. B.	Great improvement

No.	Name.	Age.	Physical signs.	Larynx	Sputum.	Result.
82	Emmanuel G.	21	Slight d. and subcrep. at left apex; mitral systolic murmur	—	T. B.	Signs of phthisis became more marked.
83	Robert S.	25	Slight d. right apex; doubtful signs of excavation; no râles	—	T. B.	No change in signs; lost sight of.
84	Louisa H.	27	Slight d. and weak breathing at left apex; obscure râles on cough	—	T. B.	Signs of phthisis became well marked
(c)						
85	*John W.	50	Consolidation of left base; thickened pleura at right base	—	T. B.	Death. P.M.—Symmetrical basic tubercular phthisis.
86	Geo. P.	21	Diffused crep. both sides; d. over left back; cavernous signs at base	—	T. B.	Death. P.M.—Phthisis originating at left lower lobe. Lost sight of.
87	Matilda D.	16	Contraction of right side; d. all over back; cavernous signs at base	—	T. B.	Lost sight of.
88	Geo. C.	20	Cavernous signs at right base; upper parts free	—	T. B.	Lost sight of.
89	*Oliver D.	18	D., tubular breathing, pectoriloquy, and coarse râles at right base	—	T. B.	Signs persistent. Improvement.
90	Geo. T. H.	42	Thickening of pleura and consolidation of lower part of right lung	—	T. B.	Signs persistent. Slight improvement.
91	Fanny S.	22	D., and cavernous signs over right lower lobe	—	T. B.	Signs persistent. Chronic course.
92	*Sydney B.	10	Flattening at right base; slight d. over right back; coarse râles and weak breathing. Diagnosis: Bronchiectasis; secondary tuberculosis.	—	T. B.	Cavernous signs appeared at right apex. Gradual failure. Death at patient's home. No P.M.
93	Allan L.	6	Diffused bubbling râles on both sides; d. at both bases with tubular breathing and pectoriloquy. Diagnosis: Bronchiectasis.	—	Very foul; No T. B.	Extreme foetor of breath. Irregular pyrexia. Death. P.M.—Diffuse bronchiectasis. Cirrhosis at bases. No tuberculosis.

No.	Name.	Age.	Physical signs.	Larynx.	Sputum.	Result.
94	John R.	30	Scattered râles ; d. and cavernous signs at both bases. Diagnosis : Bronchiectasis	—	Very foul ; No T. B.	Fœtor of breath. Pyrexia. Death. P.M.—Diffuse bronchiectasis. No tuberculosis.
95	Jas. W.	32	D. and cavernous signs at left base. Diagnosis : Cirrhosis and bronchiectasis	—	Very foul ; No T. B.	Fœtor of breath. Pyrexia. Septic pleurisy. Death. P.M.—Cirrhosis, bronchiectasis, excavation of left lung.
96	Richard N.	35	D. and cavernous signs over left upper lobe and base. Diagnosis : Bronchiectasis	—	Very foul ; No T. B.	Fœtor of breath. Death under chloroform during attempt to tap basic cavity. P.M.—Cirrhosis, bronchiectasis, excavation in in both lungs.
97	Henry W.	38	D. and cavernous signs at left base ; crep. at apex. Diagnosis : bronchiectasis	—	Very foul. No T. B.	Pyæmia ; death. P.M.—Cirrhosis, bronchiectasis and excavation of left lung. No tuberculosis.
98	James K.	32	Cavernous signs at left base. Diagnosis : bronchiectasis	—	Foul. No T. B.	Death. P.M.—Cirrhosis and bronchiectasis. No tuberculosis.
99	Wm. C.	38	Cavernous signs at left base ; harsh breathing at apex. Diagnosis : bronchiectasis	—	Foul. No T. B.	Death from hæmoptysis. P.M.—Cirrhosis and bronchiectasis. Large cavities in left lung, one of which contained a ruptured aneurism.
100	Chas. P.	19	Slight retraction at left base, with d., and signs of consolidation. Diagnosis : cirrhosis and bronchiectasis	—	Profuse. Not foul. No T. B.	Death from uræmia. P.M.—Cirrhosis and bronchiectasis. No tuberculosis. Parenchymatous nephritis.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 395.)

ON THE RELATIONS  
BETWEEN THE  
FUNCTION, STRUCTURE, ORIGIN, AND DISTRIBUTION  
OF THE NERVE-FIBRES  
WHICH COMPOSE THE  
SPINAL AND CRANIAL NERVES.  
BEING THE  
MARSHALL HALL PRIZE ORATION.

BY  
WALTER HOLBROOK GASKELL, M.D., F.R.S.

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Received May 24th—Read May 24th, 1888.

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IN order to comply with the request of the Royal Medical and Chirurgical Society to give some account of those researches for which they have awarded me the Marshall Hall Prize, I have, after consideration, determined to sketch out as rapidly as possible the work which I have done in the order of my investigations, thinking it might interest you to see the manner in which one research led to another.

In 1881 I was enabled, by the use of a new method, to show that the vagus nerve not only inhibited the rate and force of the contractions of the heart in the frog, but also was able to accelerate the rate, and to augment in the most marked manner the strength, of the contractions of the heart.

These observations led to the discovery that the vagus nerve of the frog was in reality the vago-sympathetic, that the vagus fibres proper were inhibitory, and that the sympathetic fibres not only caused the acceleration and augmentation observed but also that they reached the heart by way of the annulus of Vieussens in precisely the same manner as in the case of the mammal. I was enabled further to show that in the cold-blooded tortoise and crocodile the heart is also supplied by two nerves of opposite function, having the same course as in the mammal.

Further, my investigations convinced me that the inhibitory fibres as well as the augmentor fibres retain the same function up to their termination in the cardiac muscle, and that therefore inhibition is something taking place in the muscle itself and cannot be explained by any theories of interference of nerve action outside the muscle.

Again, taking into account not only the primary effect but also the more lasting after-effect of the stimulation of these two opposing kinds of nerve fibres, I came to the conclusion that the process of inhibition was bound up with changes in the muscle of a beneficial nature to the further action of that muscle, while the action of the augmentor nerve resembled rather the action of a motor nerve and caused an exhaustion of the muscular activity. I concluded therefore that inhibition of contraction is a symptom of the action of an anabolic nerve, *i. e.* a nerve which brings about constructive metabolism just as much as contraction or augmentation of a contraction is a symptom of the action of a katabolic nerve, *i. e.* a nerve which causes a destructive metabolism.

In connection with this conclusion the consideration of the nerve supply of the rest of the vascular system, of the muscular tissue of the alimentary tract, and of the glands, led me to suggest that all tissues are supplied with anabolic and katabolic nerves, so that the efferent nerves and their centres of origin in the central nervous system are divisible into two great anabolic and katabolic nerve systems.

With this suggestion present in my mind I was investi-

gating under a dissecting lens the course of the sympathetic cardiac fibres in the tortoise and was much struck with the difference of appearance between them and the vagus fibres going to the heart. I promptly, therefore, looked at osmic preparations of the same nerves in the dog and rabbit, and found, to my delight, that invariably the augmentor fibres between the annulus of Vieussens and the heart were entirely composed of non-medullated nerve fibres, while the vagus cardiac fibres were all small strongly medullated fibres.

Here, then, was an anatomical difference between anabolic and katabolic nerves which immediately led to the question,—Does this distinction hold good along the whole length of their course?—thus leading to the investigation of the structure of the rami communicantes and of the roots of the spinal nerves.

Now, it was known that the accelerator fibres for the heart passed out of the spinal cord in the anterior roots, and, indeed, I myself had proved that they passed out in the anterior root of the third spinal nerve of the frog. I therefore examined the structure of the anterior roots of the dog in the upper dorsal and lower cervical region and found that they were absolutely free from non-medullated fibres, so that these accelerator nerves did not leave the spinal cord as non-medullated but as medullated fibres. At the same time I was struck with the difference in structure between the anterior roots of the thoracic and cervical region, the former, as pointed out by Reissner, containing groups of very fine medullated nerve fibres, varying in size from one  $1.8\ \mu$ — $3.6\ \mu$ , which were absent in the latter roots.

Further investigation showed that these fine, medullated fibres of the anterior roots formed the bulk of the fibres contained in the white rami communicantes, so that the course of the accelerator nerves was now evident; clearly they left the central nervous system as fine medullated fibres, passed to the ganglia of the sympathetic chain, along the white rami communicantes as fine medullated fibres,



lost their medullary sheath in the sympathetic ganglia, and passed to the heart as non-medullated fibres. The anatomical difference, therefore, between the anabolic and katabolic nerves of the heart consists simply in the place where the medullary sheath is lost; they both leave the central nervous system as nerve fibres of very similar histological character.

This connection between the structure of the anterior root and of the white ramus communicans led me to a detailed investigation of the anterior roots and rami communicantes along the whole length of the nervous system, and I was enabled to show that, throughout, the presence of white rami communicantes was associated with the presence of these groups of very fine, medullated fibres in the corresponding anterior roots, so that where they were absent the rami communicantes were grey, *i. e.* composed almost entirely of non-medullated fibres, and therefore peripheral in direction, going, in fact, to supply the blood-vessels of the district supplied by the spinal nerves in question and of the membranes of the cord; while, on the other hand, where they were present both a white and a grey ramus existed.

The simultaneous existence of white rami communicantes and of these groups of fine, medullated fibres in the anterior roots was found to be limited to the region of the cord comprised between the second thoracic and the second lumbar nerves inclusive.

Further, I argued, that if in other parts of the central nervous system groups of fine, medullated fibres appeared in the anterior roots, then, even although no evident white rami communicantes were present in connection with such roots, we might expect to find something formed strictly homologous to such white rami communicantes. Such I found to be the case. The anterior roots of the second and third sacral nerves contain numbers of these smaller visceral nerve fibres which pass out to form the *nervi erigentes* and pass into the ganglia of the hypogastric plexus; and in the upper cervical region another similar group is found in the

upper roots of the spinal accessory nerve, which pass out to help form the internal branch of the spinal accessory and enter into the *ganglion trunci vagi*.

I came therefore to the conclusion that it was better no longer to speak of rami communicantes, and of the sympathetic system, but rather to use the term of the morphologist and to speak only of rami viscerales, and it was evident that such visceral nerves did not pass equally from all parts of the spinal cord of the dog, but that they formed three marked outflows:

1. A cervico-cranial outflow in connection with the vagus and spinal accessory nerves;
2. A thoracic outflow; and
3. A sacral outflow.

This marked and striking inequality in the origin of the visceral nerves led immediately to a number of other considerations.

In the first place, the inequality appeared to be clearly due to the interposition of the plexuses for the anterior and posterior extremities; for these plexuses fill up the gaps between the three visceral outflows.

In the second place, the limits of the outflows correspond so closely with the limits of Clarke's column of cells as to force one irresistibly to the conclusion that this group of cells is connected with visceral nerves.

In the third place, an explanation is at once given of the extraordinary round-about course which the constrictor nerves of the blood-vessels and the so-called sympathetic glandular nerves take to reach their destination in the various parts of the body; for such nerves are apparently confined to the thoracic outflow, and losing their medullary sheath in the ganglia of the so-called main sympathetic chain, pass in all directions to their destination as non-medullated fibres.

Further, I was able to point out how this appearance and distribution of small medullated fibres corresponded to known facts as to the distribution of the nerves governing the muscles of the alimentary tract, how the known cases

of vaso-dilator nerves and the secretory nerves of the glands were all characterised by the fineness of the calibre of their fibres and the presence of ganglion-cells on their course; that in fact a large class of nerves existed of various functions which might all be called visceral or splanchnic nerves, all characterised by the fineness of their fibres, and by the peculiarity that although efferent in function they were in connection with ganglia in some part of their course.

The result of the investigation therefore up to this point led me directly to regard the sympathetic and homologous ganglia as the motor or efferent ganglia of these visceral fibres, so that instead of the old conception of two nervous systems which interchanged fibres with each other I would substitute the following definition of the nerves belonging to a spinal segment:—

A spinal nerve is composed of an anterior and posterior root, both of which are ganglionated, the difference between the two roots being that the ganglion of the afferent root is always stationary in position, being situated near the exit of the nerves from the central nervous system, while that of the efferent root is vagrant and has travelled away to various distances from the central nervous system. Moreover, these vagrant efferent ganglia are connected with the specially fine-fibred visceral efferent nerves.

This conception that fully-formed spinal nerves possessed motor ganglia as well as sensory ganglia appeared at once to throw light upon the vexed question of the relationship of the cranial to the spinal nerves, and to suggest the removal of the difficulty, so frequently noticed in morphological works, that a nerve which corresponded apparently to a posterior root should contain fibres which were clearly motor in function.

I determined therefore to compare the structure of the cranial with the spinal nerves and see how far they corresponded.

The first point which struck me was that these fine ganglionated efferent visceral nerve fibres appeared to have a more or less special relationship to a group of large-

fibred motor nerves, which were especially concerned with the function of respiration and deglutition, such as the external branch of the spinal accessory, the pharyngeal and laryngeal motor nerves of the vagus group, the nerves of the muscles of expression and deglutition; for in close connection with all these nerves are found groups of small medullated fibres, which are efferent in function and pass to various ganglia. It was possible therefore, that some close connection existed between these small-fibred efferent nerves and these large motor nerves which corresponded closely with Bell's group of respiratory nerves.

The connection appeared to me to be clearly pointed out by van Wijhe's researches on the mesoderm segments in the head of Selachians. For he has pointed out that a double segmentation is plainly visible in the head. On the one hand, a dorsal segmentation into somites from which are formed a series of muscle-plates which give rise to the muscles innervated by the third, fourth, sixth, and twelfth nerves, and, on the other hand, a ventral segmentation in connection with the formation of visceral and branchial clefts forming the lateral plates of mesoblast, *i. e.* the mesoblast surrounding the walls of the clefts, which give rise to the muscles of mastication, the facial muscles, and the muscles of deglutition and respiration; muscles supplied by the fifth, seventh, ninth, and tenth nerves.

In other words, if we use the terms somatic and splanchnic to denote the dorsal and ventral segments respectively, we can look upon the nerves of the body as divisible into two corresponding groups, *viz.* somatic and splanchnic groups, and can therefore define a spinal nerve as composed of:

1. A posterior root with a stationary ganglion, into which both somatic and splanchnic afferent fibres pass.

2. An anterior root, composed of (a) a large-fibred somatic and splanchnic non-ganglionated portion, and (b) a small-fibred ganglionated splanchnic portion, of which the ganglion is vagrant.

By somatic meaning that the nerves supply parts derived

from the epiblast and from the mesoblastic somites; by splanchnic meaning nerves which supply parts derived from the hypoblast and from the rest of the mesoblast.

The examination of the evidence as to the origin of the cranial and spinal nerves in the central nervous system which is given in the text-books of human anatomy, fully confirmed the existence of such a division of the nervous system, for the arrangement of the nerve cell groups in the spinal cord appear naturally to fall into two groups, one of which is in connection with somatic and the other with splanchnic nerve fibres, and it appears to me that the nerve cells of the somatic nerves are situated in the out-lying horns of the grey matter, while the nerve cells of the splanchnic nerves form a group in the neighbourhood of the central canal.

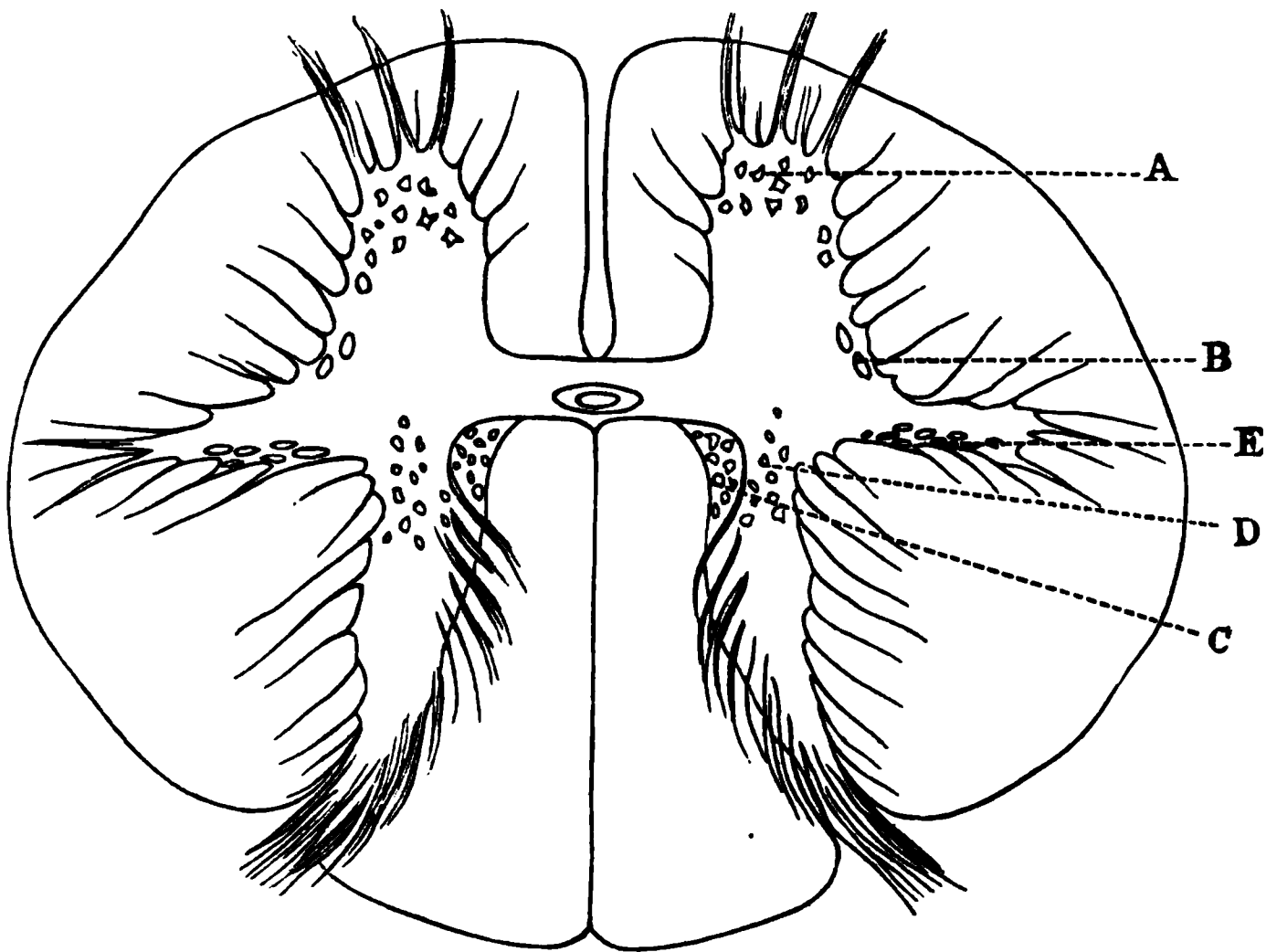
Taking into consideration the corresponding groups in connection with the cranial nerves, we have five groups for investigation :

Firstly, the cells of the anterior horn (A in Fig.), which are known to give origin to the large motor nerves, and which are continued cranial-wards as the nuclei of the twelfth, sixth, fourth, and third nerves. These are clearly connected with efferent somatic nerves.

Secondly, three separate groups of cells (C, D, E, in Fig.), viz. Clarke's column, the solitary cells of Schwalbe, and the cells of the intermedio-lateral tract, which all appear to be connected with splanchnic nerves, for the cells of Clarke's column coincide with the outflow of visceral nerves in the spinal cord, form parts of the vagus nucleus, and apparently are continued upwards as the cells of the *locus coeruleus*. The solitary cells of Schwalbe appear to increase largely in number as the vagus nucleus is approached, for which reason they also must be looked on as splanchnic.

The cells of the intermediate lateral tract as pictured by Schwalbe are well defined only in the thoracic region corresponding to the outflow of vaso-motor nerves, and if they exist in the cranial region they may perhaps constitute that group of cells known as the antero-lateral

nucleus in the medulla oblongata which is defined by the observations of Ludwig and his pupils as the vaso-motor centre.



Section of spinal cord in the dorsal region  
(slightly modified from Schwalbe).

Thirdly, it is significant that the centres of origin for the motor nerves of the muscles of mastication, deglutition, of expression, of the pharynx and the larynx, should, as is well known, form a well-defined consecutive series in the central nervous system separate from the groups which give origin to the somatic motor nerves; in fact, separate centres of origin for the non-ganglionated efferent nerves of the splanchnic system corresponding to the separate origin of the muscles which these nerves supply.

In the spinal cord this group of nerve cells forms a part of the lateral group of motor cells in the cervical region, and it is doubtless connected with some at all events of the motor fibres of the spinal accessory and with the phrenic nerves.

It is not apparently the same as the smaller cells of the

*tractus intermedio-lateralis*, and it appears to me suggestive in consideration of the embryonic position of the muscles supplied by this group that in the thoracic region a few larger cells (B in Fig.) should be found connecting the cells of the *tractus intermedio-lateralis* with the cells of the anterior horn proper.

Without speculating further into the functions of the nerves which are probably connected with these different groups of nerve cells, enough has been said to show that not only the nerves themselves but the central nervous system itself is divided, in accordance with the natural division of the body, into somatic and splanchnic segments, and seeing that such segments are only well defined in the head, it was natural to endeavour to see how far the structure of the cranial nerves threw light upon their segmental arrangement.

In connection with this question I had previously noticed a fact which is of importance in estimating the spinal character of the cranial nerves, viz. that the anterior root of a spinal nerve does not necessarily pass free from the posterior root-ganglion. In most of the nerves of such an animal as the tortoise, in the two first cervical nerves of the dog, the fibres of the anterior root pass into the root-ganglion and are mixed up with its cells, so that it is not necessary for a spinal nerve to possess an anterior root, which runs free from the root-ganglion.

We may therefore look on a cranial nerve as built up on the same plan as a spinal nerve, if it possesses a large-fibred splanchnic and somatic efferent portion, a small-fibred splanchnic efferent portion in connection with a vagrant ganglion, and a splanchnic and somatic afferent part in connection with a stationary ganglion, even although the efferent nerves do not run separately from the stationary afferent ganglion.

As yet I have not finished the investigation of the structure of the cranial nerves, but have seen enough to satisfy myself that they were originally built up on the same plan as the spinal nerves.

If we leave out of consideration the nerves of special sense, viz. the optic, olfactory, and auditory, we find that the remaining nerves fall naturally into two groups :

1. A foremost group, which in man are almost entirely efferent, viz. oculo-motor, trochlearis, abducens, motor part of trigeminal, and facial.

2. A hindmost group of nerves of mixed character, viz. glosso-pharyngeal, vagus, spinal accessory, hypoglossal, and sensory part of trigeminal.

The nerves of the first group resemble spinal nerves in their anterior roots, for they contain a large-fibred efferent portion which supplies voluntary muscles, both of the splanchnic and somatic systems, and a small-fibred efferent portion connected with vagrant ganglia. In the oculo-motor nerve these bundles of small fibres are especially well seen, for they separate out from the large fibres supplying the striated muscles of the eye, and pass into the oculo-motor ganglion, from whence they pass along the short ciliary nerves to supply the sphincter and ciliary muscles. In the trochlearis nerve groups of small fibres are also found, but as yet I have not traced them to any vagrant ganglion. In the motor part of the trigeminal the small fibres are probably in connection with the *ganglion nasale*, while in the abducens nerve it is doubtful whether such groups of small fibres exist, this nerve being probably the somatic portion of the motor part of the trigeminal. In the facial the groups of small fibres compose the main part of the *nervus intermedius*, and pass into the *ganglion geniculatum*.

They resemble them also in their posterior roots, inso-much as they possess a ganglion near the place of exit of the nerves from the central nervous system. There is, however, a great difference between the root-ganglion of these nerves and that of an ordinary spinal nerve, for in their case neither the ganglion cells nor the nerve-fibres in connection with them are any longer functional; both nerve fibres and nerve cells are phylogenetically degenerated, so that a characteristic arrangement of connective-



tissue elements is all that is left to show where the nerve-structures have formerly been (see Pl. II).

This foremost group, then, of cranial nerves is composed of at least four complete segmental nerves built up on the same type as the segmental spinal nerves, and all characterised by the peculiarity that the afferent roots with their root-ganglia have undergone degeneration. These four segmental nerves are oculo-motor, trochlearis, motor part of trigeminal together with abducens, and facial.

The nerves of the second group are also formed on the same plan as a spinal nerve; here, however, no loss of any component has occurred, but rather there is good evidence that a group of at least five segmental nerves has been split up, so that the various components which characterise fully-formed segmental nerves have become separated from each other in their exit from the central nervous system, though still arising each from its appropriate group of nerve cells in the central nervous system; a scattering which is associated with an extensive origin and an extensive distribution. The nerves of this group in fact arise partly from spinal and partly from cranial regions, and are distributed over a wide area in connection especially with the alimentary canal. The different components of the group are arranged somewhat as follows:

1. The ascending root of the trigeminal, together with the auricular branch of the vagus, contains the somatic afferent nerves. The ganglion of these nerves is mainly the Gasserian ganglion.

2. The ascending root of the glosso-pharyngeal and vagus nerves contains the splanchnic afferent fibres with the *ganglion jugulare* for its stationary root ganglion.

3. The hypoglossal and probably part of the spinal accessory form the somatic efferent portion.

4. The large motor nerves of the glosso-pharyngeal (?), vagus, and spinal accessory (in part) form the splanchnic, non-ganglionated, efferent fibres.

5. The small fibres of the glosso-pharyngeal, vagus, and spinal accessory with the *ganglion petrosum glosso-*

*pharyngei*, and *ganglion trunci vagi* for their motor ganglia form the splanchnic ganglionated efferent fibres.

Thus we see that this second group is characterised not by a loss of any component but by a splitting up of the group into its separate components, a splitting up which, in all probability, is associated with the making good of the loss of those parts which was the cause of the phylogenetic degeneration so characteristic of the first group of cranial nerves.

We see then clearly that the cranial nerves, with the exception of the nerves of special sense, are built up on the same plan as the spinal nerves, but are divisible into two groups, the meaning of which remains yet to be seen.

When we have a clear conception of the change which has occurred in the past history of the vertebrate animal by which the peculiarities of these two great groups of cranial nerves have been brought about, then we shall be able to speak clearly and definitely about the number of cranial segments; at present I can only say that I hope before long to offer an explanation of the meaning of these lost parts, which I sincerely trust will receive the same kind consideration as you have given to-night to the somewhat speculative views which I have had the honour of laying before you.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 402.)

## DESCRIPTION OF PLATE II

On the Relations between the Function, Structure, Origin, and Distribution of the Nerve-Fibres which compose the Spinal and Cranial Nerves (WALTER HOLBROOK GASKELL, M.D., F.R.S.).

The sections are all carefully drawn from osmic acid preparations of the nerves of man.

FIG. 1.—Transverse section of rootlet of third cranial nerve to show degenerated ganglion. (Zeiss A, Oc. 4.)

FIG. 2.—Three transverse sections of fourth cranial nerve (Zeiss A, Oc. 2) :

- a. Section near exit of nerve from valve of Vieussens, showing degenerated tissue arranged so as to form a sheath around the functional medullated nerve-fibres.
- b. Section of nerve farther away from point of exit, showing the formation of the degenerated ganglion.
- c. Section of nerve peripheral to the ganglion. The degenerated tissue has almost entirely disappeared.

FIG. 3.—Transverse section of seventh cranial nerve to show degenerated ganglion. (Zeiss A, Oc. 2.)

FIG. 4.—Longitudinal section of a rootlet of third cranial nerve to show the structure of the degenerated material. (Zeiss D, Oc. 2.)

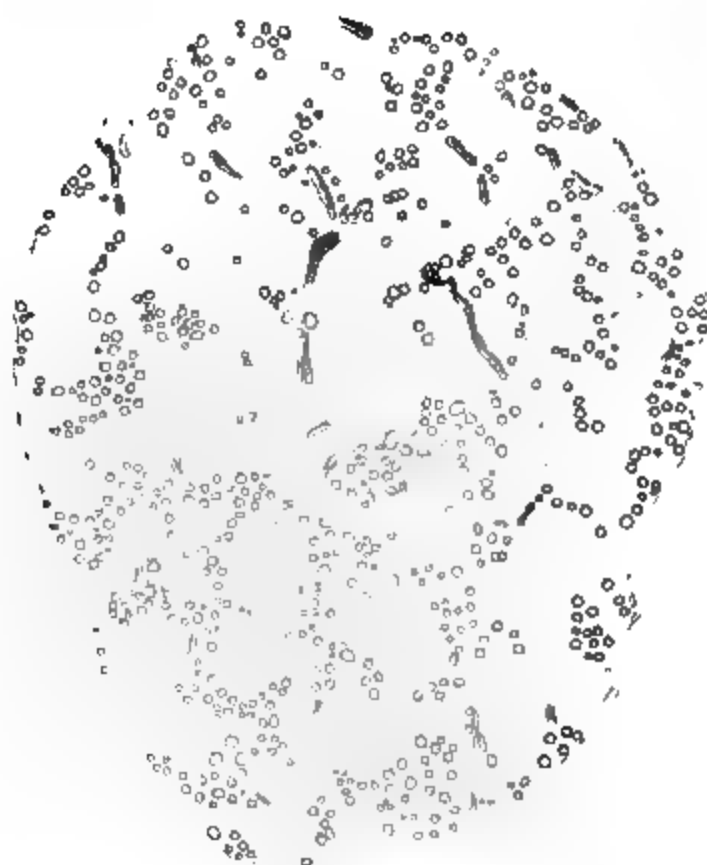


Fig 1

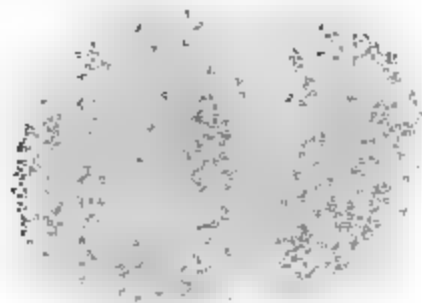


Fig 2 a



Fig 2 b

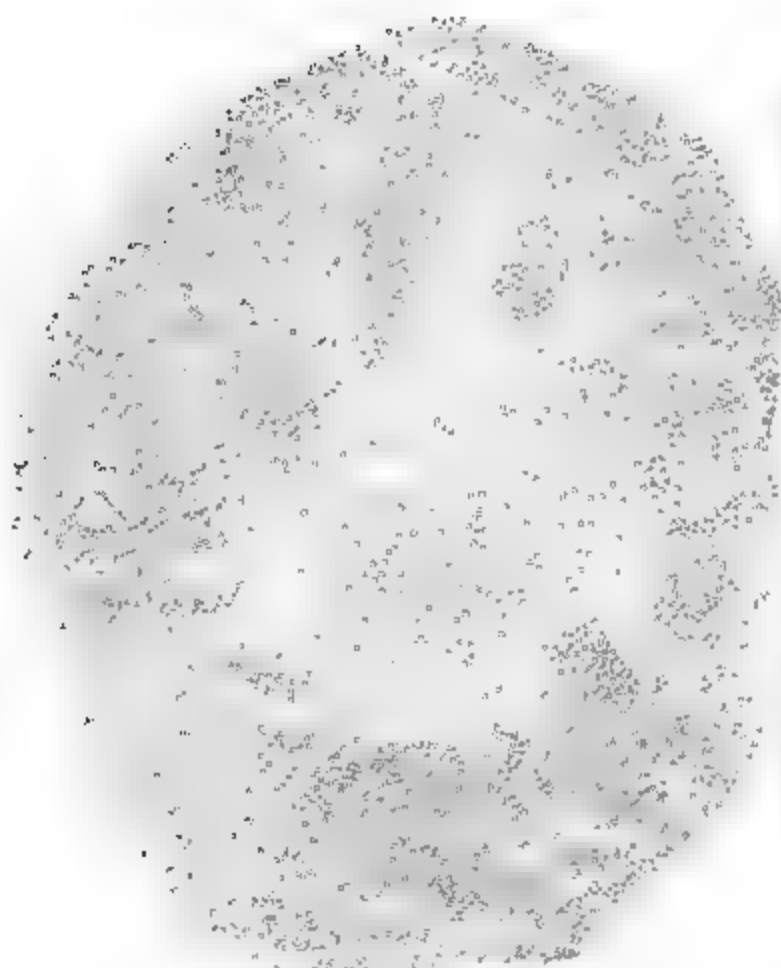


Fig 3

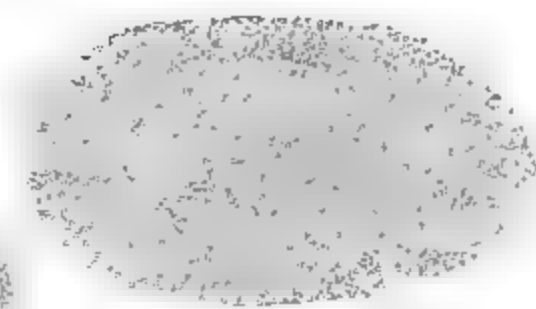


Fig 4

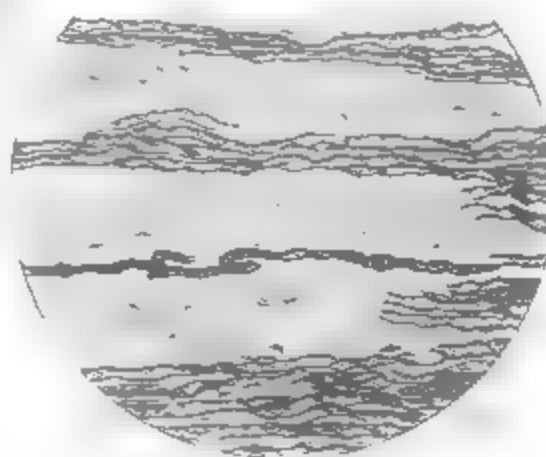


Fig 4



A C A S E  
OF  
TUMOUR OF THE SPINAL CORD.  
REMOVAL; RECOVERY.

BY  
W. R. GOWERS, M.D., F.R.S.,  
AND  
VICTOR HORSLEY, B.S., F.R.S.

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Received March 8th—Read June 12th, 1888.

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*Medical History of the Case, by Dr. GOWERS.*

CAPT. G—, æt. 42, had good health until the year 1884. There was no history of syphilis. During 1883 and 1884 he endured much mental anxiety, and in the latter year he had a considerable mental shock—his wife was knocked down and run over in his presence, and he was able to save himself from a similar fate only by suddenly throwing himself backwards. Soon afterwards he began to suffer from a dull pain across the lower part of the back, which he thought was due to the strain of the accident. This pain passed away in the course of a few weeks and did not return. In June, 1884, he first felt a peculiar pain that was the most

prominent symptom during the early part of his illness. It was localised in a spot beneath the lower part of the left scapula. This pain commenced suddenly one day while he was walking, and was continuous and severe for about a month. It was increased by active exertion and by the jolting of a carriage. Repeated examination failed to reveal any cause for it. After a time it became less, but was felt occasionally through the autumn and winter. By the spring it had all but ceased, and he was asked to go out to China on business. Before undertaking the journey he consulted a physician in London, who pronounced the pain to be an intercostal neuralgia and suggested that the voyage would probably do good. While Capt. G— was in the train, on the way to Brindisi, the pain returned in severe degree, at the same place, and of the same character. During the voyage it continued, varying in severity, but when he reached China it was so intense, and was so much increased by movement, that he could scarcely walk. A German doctor at Shanghai, after a course of Turkish baths had been tried without benefit, expressed the opinion that an aneurism was the cause of the pain. Digitalis and iodide of potassium were given, and the latter was increased to large doses by two English practitioners at Shanghai, who doubted, however, whether there was an aneurism. The pulse became curiously variable, changing from 120 in the morning to 75 in the afternoon. The pain continued, and some fainting attacks occurred, one of which was thought to be possibly epileptic in character. In October, 1885, still suffering much and very prostrate, he left China for England. During the voyage he improved in health, the faintings ceased, and the pain lessened, so that in December, 1885, he could walk a little. Walking had been interfered with only by the pain. Other physicians were consulted, and the rest of the winter was passed in the South of France. The improvement continued, and by the spring of 1886 he was so much better that he went on to Constantinople on business. While there the pain almost ceased. He returned to England in the middle of the summer, and, as the pain

was still felt a little at times, he consulted other physicians and by them was sent to Aix-la-Chapelle. While he was there the pain returned in great severity and morphia was injected. In September, 1886, he returned to England, and the pain was then very severe, and, as before, was increased by movement so that he was again scarcely able to walk. The morphia was stopped and blisters applied. An aneurism was again suggested as the probable cause of the symptoms, and the use of morphia was resumed for a time, but was again discontinued at the wish of the patient himself. He became irritable; the continued pain seemed to lessen his power of self-control. So marked, indeed, was his mental state that the question was seriously raised whether he was quite sane, and whether this mysterious pain was anything like as severe as he described. He continued in this condition till the end of the year. In February, 1887, he again came to London for advice and consulted two physicians, who expressed the opinion that there was no organic disease and advised him to go abroad. During February and March there came on distinct loss of power in the legs. The left leg first became weak and a few weeks afterwards the right. In April he went abroad, and remained away from England for two months. During this time the weakness increased to complete loss of power, sensation became impaired, and the urine was retained in the bladder. Still the mental peculiarities were so conspicuous to those around him that fresh doubts were felt as to the reality of his symptoms, and it was suggested by someone that he should be put through a course of the Weir-Mitchell treatment. Before this step was taken another opinion was thought desirable, and the patient was brought to London for the purpose on June 4th. I saw him on the following day, in consultation with Dr. Percy Kidd, who was connected with the patient but had not had anything to do with the previous treatment.

The condition then presented by Captain G— was that characteristic of grave organic disease of the dorsal region of the spinal cord. There was absolute palsy of the legs,



and cutaneous sensibility of all kinds was lost as high as the ensiform cartilage. At and just above this level, that is, in the region of the sixth and seventh intercostal nerves, he complained of severe pain around the chest, much more severe on the left side than on the right, and increased to evident agony on any movement. The legs from time to time became rigid in extensor spasm, and a clonus could be obtained with great readiness in the muscles of the calf and front of the thigh. The paroxysms of spasm involved also the muscles of the abdomen. The bladder was distended, and the urine that was drawn off contained pus. There was no irregularity of the vertebral column, nor could tenderness be discovered in any part. No trace of pulsation could be felt in its vicinity, and no murmur could be heard on auscultation. The thoracic organs seemed healthy, and both lungs were equally filled with air.

The development of complete paraplegia, which had taken place during the preceding four months, rendered the diagnosis, up to a certain point, a simple matter. The symptoms were those characteristic of a transverse lesion of the cord a little above the middle of the dorsal region. The gradual onset of the paralysis, the affection of one leg before the other, and the long-preceding signs of nerve irritation at the level of the lesion, made it practically certain that the spinal cord was damaged by compression and that the cause of the pressure was outside the cord itself. Caries of the spine was excluded by the absence of any irregularity of the spines or tenderness, taken in conjunction with the long duration of the symptoms. The diagnosis lay between an aneurism eroding the vertebræ and compressing the cord, a growth springing from the bones of the spine, and an intraspinal tumour within the canal, but outside the cord itself. Although aneurism could not be completely excluded, the absence of any of the characteristic physical signs of aneurism, and the absence of any indication of weakening of the spinal column, made this cause of compression far less probable than one of the two others. The distinction of a

tumour of the bones from one within the canal can only be a matter of certainty when the enlargement of the bones, caused by the former, can be felt. In other cases the diagnosis can only be a matter of probability, and often of very low probability. A growth backwards from the bodies of the vertebræ may cause symptoms undistinguishable from those due to a tumour springing from the membranes. In this case, however, the symptom of longest duration, the pain, pointed to irritation of the posterior roots on the left side, and therefore to a lateral position of the growth, and the affection of the left leg before the right had the same significance. A growth from the bone on one side of the cord would be more likely to cause recognisable enlargement of the parts, than would one springing from the bodies of the vertebræ, and the absence of such enlargement in this case was therefore somewhat in favour of the growth being altogether within the canal.

The course of the symptoms, coupled with the inutility of iodide of potassium, precluded the supposition that the disease was syphilitic. The long duration of the symptoms in slight degree was in favour of the non-malignant character of any growth that might exist.

In a description of tumours within the spinal canal,<sup>1</sup> I had previously suggested that the removal of spinal meningeal growths would be not only practicable but actually a less formidable operation than the removal of intracranial tumours. In this case the patient and his friends were exceedingly anxious that something should, if possible, be attempted. An operation gave a chance, the only chance, of cure. If the tumour should turn out to be one that could not be extirpated, it was possible that the removal of an arch, or the division of nerve-roots passing into the growth, might lessen the sufferings of the patient. If nothing were done, death after months of intense suffering was inevitable.

Sir William Jenner saw the patient with Dr. Percy Kidd and myself, and concurred in the probable diagnosis of a growth. The question of an operation was submitted to

<sup>1</sup> 'Manual of Diseases of the Nervous System,' vol. i, p. 432.

him and received his sanction, provided the patient himself clearly understood the nature of the operation and that a perfectly successful result was not more than a possibility. Capt. G— was, however, only too anxious to submit to anything that held out the faintest hope of relief. Accordingly Mr. Horsley was asked to see the patient, and, if he saw fit, to operate.

*Surgical History of the Case, by Mr. VICTOR HORSLEY.*

I saw Captain G— on the 9th of June, 1887, at 1 p.m. The patient was half sitting up, complaining of paroxysms of very great pain in the lower limbs and abdomen, the former being completely paralysed and frequently flexed in clonic spasm, the pain accompanying which was so severe as to cause the patient to cry out. On careful examination of his spine, there appeared no undue prominence of any vertebra, and the only abnormality detected was tenderness on pressure to the left side of the sixth dorsal spine. This was very constant though slight; on movement the patient complained of a sensation of weakness (rather than pain) referred to the middle of the dorsal region, but such movement did not seem to start the spasm in the legs by interference with, or pressure upon, the spinal cord. He was very loth to move because it necessitated voluntary change of position of his legs, movement of any of the joints of which was liable to bring on a severe paroxysm of painful flexion. In addition to the complete loss of motor power just noted, there was loss of tactile sensibility as high as, and involving the destruction of, the fifth dorsal nerve. There was some doubtful diminution of sensibility in the left fourth intercostal space, but this could not be satisfactorily demonstrated when I saw the patient.<sup>1</sup> On the right side the insensibility was limited to the fifth interspace. The anæsthesia was complete for all kinds of stimulus.

<sup>1</sup> This slight affection of the left fourth nerve was nevertheless of great diagnostic importance as the sequel of the operation shows (see p. 385).

There was complete loss of power over the bladder and rectum, and catheterisation had been found difficult with a metal instrument on account of the severity of the urethral spasm thus excited. (After the operation, when the spasmodic condition was equally severe, the passage of a soft rubber catheter was unattended by this trouble.)

The morning temperature during the week preceding the operation varied from  $97.4^{\circ}$  to  $99.2^{\circ}$ , and the evening temperature between  $99^{\circ}$  and  $99.4^{\circ}$ .

For the history and present state see the foregoing description by Dr. Gowers.

*Operation.*—June 9th, 3.30 p.m. Present: Drs. Gowers, Percy Kidd, and Edmunds. Mr. White anæsthetised the patient with ether while he was lying in the semi-prone position on the right side, and I was kindly assisted by Mr. Stedman and Mr. Ballance. The skin was shaved and thoroughly cleaned with ether and 5 per cent. carbolic acid solution, the spray was used throughout the operation, and the instruments and sponges were kept in 5 per cent. carbolic solution. Free incision was then made in the middle line through the skin and the subcutaneous tissues extending from the third dorsal spine to the seventh. The deep fascia and tendinous attachments of the muscles were then cut from the spines and a transverse cut was carried outwards from the spines over the spinous muscles through the vertebral aponeurosis, so as to prevent all tension on the sides of the wound. (See Remarks.) Vessels bled freely by the sides of, and between, the spinous processes, and were secured with Wells's forceps. The muscles were then completely detached from the spinous processes, from the laminæ, and from the mesial aspect of the transverse processes. This was done in a way which I shall refer to later, namely, by free use of the knife, and subsequently blocking the wound with sponges, while the same procedure was carried out on the other side of the spinal column. The sides of the wound being now strongly retracted and most of the vessels ligatured, the spines and laminæ could be seen perfectly.

The fourth, fifth, and sixth dorsal spines were then cut off close to their bases with powerful bone forceps, the laminal arch of the fifth vertebra was then trephined with a three quarters of an inch trephine, the pin being placed in the middle line. The bone was very hard and tough and one sixteenth of an inch thick. The rest of the laminæ were then removed with a bone forceps and knife, the ligamenta subflava giving much trouble owing to their toughness. The laminal arches of the fourth, fifth, and sixth vertebræ being thus cleared away, the dura mater was easily exposed by an incision in the middle line through the fat covering it. This fat, being pressed aside, shrank and showed the dura mater of a normal appearance, colour, and tension. Nothing very abnormal was then observed, save that on the left side the dura mater was distinctly pressed nearer to the bony wall of the neural canal. This, of course, was due to the fact that the tumour lay on the left side of the cord, and consequently pressed the dura mater on that side closer to the vertebræ. The wound being practically bloodless, the dura mater was slit open in the middle line with a knife and dissecting forceps. The cerebro-spinal fluid escaped freely, but not with any undue pressure to signify pathological tension. The spinal cord was now exposed for about two inches and appeared to be perfectly natural in colour and density; moreover, the vessels coursing on its surface were in every respect normal.

It will now be readily understood that the upper part of the roots of the sixth nerve and the whole course of the fifth nerve on each side from the spinal cord to the intra-vertebral foramen was completely exposed. Examination of the spinal cord on all sides with the finger and cautiously with an aneurism needle failed to reveal anything abnormal. Another lamina was removed at each end of the wound, the dura mater as before slit up, and the cord still further exposed, but still nothing pathological was discovered. At this juncture it appeared as if sufficient had been done, but I was very unwilling to leave the matter undecided, and my friend Mr. Ballance being strongly of the opinion that

further exposure of the cord was indicated, I determined to go further if the state of the patient warranted me in so doing. Finding that his pulse was very strong, and that there would be no difficulty whatever in the anæsthetisation, I removed another lamina at the upper part of the incision. On opening the dura mater I saw on the left side of the subdural cavity a round, dark, bluish mass about three millimetres in diameter, resting upon the left lateral column and posterior root-zone of the spinal cord. I recognised it at once to be the lower end of a new growth, and therefore quickly cut away the major part of the lamina next above. This enabled me to see almost the whole extent of the tumour when the dura mater was divided. It was an oval or almond-shaped body of a dark, bluish-red colour, resting upon, and attached at its lower extremity to, the highest root of the left fourth dorsal nerve,<sup>1</sup> just where the posterior nerve-roots were gathered together in one trunk. On palpation the tumour markedly fluctuated. Above, it extended as far as the third dorsal nerve to which it appeared to be loosely attached by connective tissue, evidently a fold of the arachnoid. The tumour occupied exactly the position of the point of the ligamentum denticulatum, being jammed between the dura mater and the left side of the spinal cord. The pia mater and the arachnoidal sheath of the spinal cord evidently passed continuously from the cord over the surface of the tumour, forming a kind of capsule on its upper surface. At the same time it seemed as if the tumour could be pressed away from the spinal cord, so as to give the idea of its not actually invading the substance of the cord. I therefore made an incision through the pia mater sheath of the spinal cord, and then found that I could easily dissect the tumour from the surface of the cord, lifting it out of the deep bed which it had formed for itself in the lateral column of the cord. It was easily detached above by cutting through the loose tissue before described. Below, as it was firmly adherent

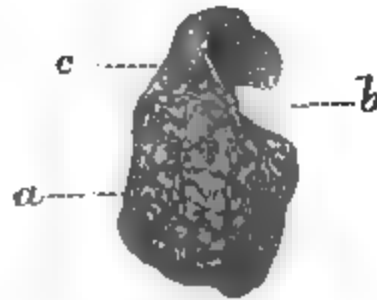
<sup>1</sup> The growth was found by measurement (at the operation) to be situated four inches above the level of complete anæsthesia.

to the fourth dorsal nerve, and as that nerve was of course of insignificant importance, I cut away the portion of nerve adherent to the growth. The outer border of the tumour was bathed in the cerebro-spinal fluid, and so required no dissection, but in removing the growth its inner surface, formerly of course in close contact with the cord, apparently gave way and some turbid serous fluid escaped, this reducing the volume of the tumour to about three fifths of its former size. (For description of the tumour *vide infra*.)

The cavity left by the removal of the growth was of course for the most part simply the subdural space, but the spinal cord was evidently greatly damaged by the pressure of the growth. The lateral column was so depressed or notched, so to speak, that the bottom of the groove in it nearly reached the middle line of the cord. It seemed likely, therefore, that most of the fibres in this column would be completely destroyed, moreover, there was evidently no resiliency in the damaged cord, for during the time that it was under observation the bottom of this pit showed no tendency whatever to rise. The surrounding adhesions of loose connective tissue oozed rather freely, but gentle pressure with a fragment of sponge for a few minutes soon arrested this bleeding. The cord and subdural space was then carefully sponged with 5 per cent. carbolic acid solution and freed from blood-clot. The edges of the long incision in the dura mater were then approximated (incision fully four inches) and laid in position but not sutured at all. The few remaining vessels were ligatured, and the sides of the wound brought together by strong silk sutures passed vertically with curved needles through almost the whole thickness of the side of the wound, and at a distance of about half an inch from the border. These sutures were placed at distances of about one inch, and on being tied firmly were found to readily approximate the two sides of the cavity close to the dura mater. The edges of the skin were carefully approximated with numerous horsehair sutures, a small superficial

drainage-tube was placed at the lower extremity of the wound, and a long drainage-tube was placed vertically to the dura mater and reaching so far as that membrane at the upper end of the wound. The whole was then covered with a strip of carbolic gauze dipped in 5 per cent. carbolic solution, and a carbolic gauze dressing applied. The patient was put back to bed.

**THE TUMOUR.**—The growth, on microscopical examination, was found to be fibro-myxoma. It presents a nodular appearance, and the cavity referred to on p. 386 was found to be on the inferior and outer surface, being such a cystic space as



Photograph of the tumour, the natural size.

- a. Points to the lobulated surface of the solid portion of the tumour, this producing the excavation of the cord.
- b. Points to the open cavity in the tumour, this cavity being ruptured during the removal of the mass.
- c. Shows the fibrous capsule forming part of the inner wall of the cystic cavity, and consisting of simple connective tissue, thus contrasting with the myxomatous tissue at a.

might have resulted from a hæmorrhage. The wall of the cavity was found to be a false capsule derived from the pia mater and arachnoid. Unfortunately its contents were lost in the operation. The mass of the growth on section was pale and homogeneous, but indications of separation into nodular masses could be seen here and there. The tumour was enveloped, as already described, in a thin capsule, and consequently the parts will first be described under the headings of capsule and substance.

(1) *Capsule.*—The capsule was formed of very delicate connective tissue in which the ground substance was obviously mucinoid, and in which numerous corpuscles were



embedded. The corpuscles were (*a*) leucocytes with darkly-staining round nuclei, (*b*) connective-tissue corpuscles with feebly-staining oval nuclei. Numerous large vessels with very thin walls (those of the veins in fact showing but one or two layers of muscle-fibres at the most) coursed through the exterior of the growth.

(2) *Substance.—Stroma.*—The stroma of the growth was composed of mucinoid ground substance and trabeculæ of spindle-shaped connective-tissue corpuscles closely applied to one another.

*Parenchyma.*—These trabeculæ marked off round spaces, which were entirely occupied with myxomatous connective tissue, *i. e.* mucinous ground substance which shrank greatly in alcohol, and in which were numerous corpuscles of varied shape. The nuclei of the corpuscles were (*a*) round and darkly staining, (*b*) oval and slightly staining, (*c* and *d*) ellipsoidal and spindle-shaped, darkly staining, and (*e*) very elongated, also darkly staining. These latter were as long and slender as the nuclei of involuntary muscle-corpuscles.

(3) *Vessels.*—A very few vessels were visible in the substance of the growth, and there were very small arterioles and venules with delicate walls of adventitia.

(4) *Pigment.*—In very many parts of the sections numerous collections of hæmatoidin granules were visible. In some instances these were obviously in corpuscles. No signs of any recent hæmorrhage, *i. e.* within a year or two, could be found.

#### *Further Course of the Case.<sup>1</sup>*

June 10th, 1887, 1 a.m.—Patient restless, complaining greatly of painful spasm in the legs and bladder, with sensation of distension of latter. Urine drawn off with soft catheter. The flow of urine was of the kind characteristic of complete paralysis of the bladder. No further change. Gr.  $\frac{1}{4}$  of morphia given hypodermically.

<sup>1</sup> Owing to the length of the case it will be best to give as far as possible succinct *résumés* of the course of each leading symptom, &c.

9 a.m., temperature  $97.6^{\circ}$  F. (It will be best stated here that the temperature throughout never reached  $100^{\circ}$  F., the highest recorded being  $99.8^{\circ}$  at 4 p.m. on June 11th, *i. e.* forty-eight hours after the operation. The temperature therefore will not be mentioned again, it being only needful to explain that its fluctuations were those of health, *viz.* low in the early morning and a little higher in the late afternoon.) Wound dressed, looked perfectly quiet. Tubes blocked with clot; cleared and replaced. Considerable amount of bloody serous discharge and cerebro-spinal fluid in the dressing.

On turning the patient over, a proceeding which was always difficult on account of the very severe pain in the abdomen and the left lower limb more especially, there was found an erythematous raised patch on the left side of the sacrum, but extending also across the middle line to the right side. The patch was about four inches broad and about three inches in vertical length on the left side, but only one inch on the right side. The nurse was positive that before the operation there was no such decubitus, and none was observed at the operation. This patch was immediately protected with boracic ointment spread on lint, and need not be again referred to, since it gradually disappeared, although on June 13th part of it in the centre of the gluteal fold appeared dusky, as though threatening necrosis of the skin. Fortunately it simply dried up.

11th.—Pulse 112. (The pulse varied very slightly after the operation, the rate gradually falling, thus on the 12th it was 108, on the 13th 95, and it varied between 90 and 100 till complete convalescence. Nothing was to be found to account for the high rate, except the pain. The patient still complained of much constant burning pain in abdomen, bladder, and limbs, in addition to which he suffered from violent painful spasm in the bladder and left leg especially. The left lower limb frequently flexed in spasm, but the right very rarely.

It need scarcely be stated that the indescribably excessive pain under which the patient laboured made examina-

tion of the limbs, &c., practically impossible, since the least touch excited the most violent clonic spasm, followed by tonic spasm, *i. e.* rigidity in extension, such spasm being agonisingly painful.

*Urine.*—Sp. gr. varied from 1024 to 1030. Strongly acid, no albumen or sugar. The urine never altered throughout, save a little in specific gravity, and therefore will also not be again referred to.

12th.—Wound dressed, uniting by first intention, though the edges looked reddish.<sup>1</sup> Discharge slightly serous, but enormous quantities of cerebro-spinal fluid escaped. The smaller (*i. e.* the lower) drainage-tube was removed, the higher left in. (This was an error; the tube should always be left out on the second day, so as to prevent the formation of a sinus along which the cerebro-spinal fluid by escaping may cause serious annoyance.)

13th.—The patient still complained of the incessant pain keeping him without sleep. Hypodermic injections of morphia in half-grain doses only gave half-hour snatches of troubled sleep, therefore draughts of chloral and bromide of potassium were given (Hyd. Chlor. gr. xv, Pot. Brom. gr. xc in the twenty-four hours, and these were continued in gradually diminishing quantities during the succeeding weeks until about the end of September).

The bowels being constipated, an enema was given with result. The urine was drawn off whenever the patient felt it distend the bladder, *i. e.* about every six hours, and when the amount of urine was usually seven ounces. It is important to note that the patient said that the distension of the bladder increased the spasm of the abdominal muscles. This spasm was tonic and never (?) relaxed until some two months after the operation.

14th.—Wound dressed, union still perfect. Dressing soaked, but this was less marked than on the 12th inst. At 6 p.m. this day, *i. e.* on the fifth day after the operation,

<sup>1</sup> I have noticed this reddish colour of the edges and suture holes in another case where the cerebro-spinal fluid rendered the wound sodden, but in which nevertheless union also occurred by the first intention.

patient passed seven ounces of urine, and again at 9.30 p.m. six ounces. The micturition, however, was not "voluntary," but purely reflex action of the just recovered lumbar centre. The patient did not know when the urine escaped, although sensation was returning.

15th.—Sensation to touch is now rapidly recovering, only the left foot being still anæsthetic. On account of the severe pain of the spasms excited by touching the limbs no further observations were made, *i. e.* as to transmission of painful impressions, of heat and cold, &c., but there is little doubt that as regards tactile and painful stimuli, sensation had returned by about the tenth day after the operation. There remained for some time a subjective sensation of heat in the left lower limb especially. Wound dressed; the remaining (the upper) drainage-tube was removed. The last time the catheter was used was 5 p.m. this day, the patient passing urine afterwards about ten times in the twenty-four hours, and about four ounces on the average each time, the rare extremes being two ounces and eight ounces.

On the 23rd, *i. e.* fourteen days after the operation, the frequency had diminished to six times in the twenty-four hours, *i. e.* normal. The constant pain was very severe and at times became excruciating, in fact the patient thought it was worse than before the operation.

16th.—The spasms by this day had gradually become restricted to the left side and lower limb, except when very severe. The cerebro-spinal fluid continued to ooze in considerable quantity through the small track of the upper drainage-tube.

22nd.—The patient steadily improved, the appetite returning, &c. This day he for the first time distinctly moved the right lower limb at the hip by "voluntary effort." On this occasion and also when (see below) the patient moved his left lower limb for the first time, he regarded the movement as only a spasm and not purposive, the muscular sense from disuse (?) being apparently deficient. (I have noted the same phenomenon in a case recently under my care in

which release by trephining of pressure on the spinal cord restored movement in the paralysed limbs.) The recovery of power spread down the limb to the foot.

At this time, however, the pain was still excessively severe at times, but was more paroxysmal, which was regarded, and rightly, as a favorable change.

July 20th.—About this date motor power returned in the left lower limb in the same manner as in the right limb, *i. e.* from the hip downwards. It is impossible to fix the date of this recovery as it was suspected for several days before the 20th, but the effort or initial spasm marred the observation, no aid forthcoming from the patient's sensations for the reason given above.

Further, the flow of cerebro-spinal fluid gradually diminished and ceased about six weeks after the operation. The arrest was aided by pressure with a pad of gauze and boracic acid over the sinus opening.

In this state, *i. e.* with recovered control of bladder and rectum, and with motion and sensation in the paralysed limbs, the patient was sent to the seaside on August 13th, 1887. The pain had gradually diminished and was confined to the left side.

A jacket consisting of a steel pelvic band from which shoulder crutches took origin, was applied to prevent possible kyphosis and to protect the cicatrix.<sup>1</sup>

Nov. 17th.—In answer to queries the patient wrote on this date, "I think I am making good progress. I take a daily turn in the garden with the aid of a couple of sticks, and also a daily drive for an hour. My back and legs are still very weak and at night I suffer a good bit of pain<sup>2</sup> and my sleep is broken, but I am in all respects much better. Though I walk with difficulty the movement of my legs is natural and tends to get better every day." At this time as from the end of July the legs had been passively moved and rubbed.

<sup>1</sup> The state of the cicatrix is shown in Plate III.

<sup>2</sup> The pain was always worse at night, and for some time before it disappeared had but very rarely occurred in the day.

Jan. 24th, 1888.—This day the patient kindly allowed the members of the Society to inspect his back, &c. The only important point noticeable now in his condition was the character of the gait, which resembled that of a man rather stiff from fatigue. He could walk three miles with ease. The scar was very firm and indeed of almost bony hardness in the site of the fifth dorsal spine and arch. The patient was practically quite free from pain and discomfort and had very greatly increased in weight and muscular development.

Feb. 21st.—Seen again to-day; the patient's rapid progress continues, the gait is notably more free and natural than when last noted. Patient about to resume his professional work.

June 6th.—Letter received this day from patient states that he is in excellent health, of which the best evidence is that he recently did a sixteen hours day's work, including much standing and walking about.

*Remarks.*—This being the first case in which a tumour involving the spinal cord has been exposed and removed, it is very advisable that a full explanation should be given of the reasons which led to the adoption of the surgical procedure above described, and I think at the same time it is worth while to look back over the literature on the subject of tumours of the membranes of the spinal cord, and to see what light may thereby be thrown on this subject.

#### *Method of Operating.*

The operation of trephining the spine has been of course known to surgery since it was suggested by Heister. It has, however, always hitherto been discussed with reference to cases of injuries of the spinal column.<sup>1</sup>

<sup>1</sup> It may be interesting here to reproduce Heister's suggestion. He says ('General System of Surgery,' 6th edit., 1757, p. 140), "But to offer the patient no assistance because we despair would seem cruel and uncharitable, therefore we must try our skill though our attempts should be in vain; in

Since it was suggested, this operation has been performed, according to 'Erichsen's Surgery,' about thirty times. Before discussing its employment in these cases it must be stated that ever since it was proposed this operation has met with the greatest opposition from some surgeons for various reasons, principally no doubt because it was performed first in the prescientific epoch of surgery, *i. e.* before the introduction of antiseptic principles by Sir Joseph Lister, and consequently it obtained much of its evil reputation from the frequency with which septic infection followed the exposure of the dura mater and the subdural space. But it has also been discarded by some on account apparently of difficulties, &c., in its performance, and in fact it is regarded by some as a very difficult as well as dangerous operation. For instance, Mr. Herbert Page, in Heath's 'Directory of Surgery,' page 134, 1881, referring to the treatment of fractures of the spine, says, "The operation of trephining the spine, proposed many years ago and adopted several times, has made no progress in surgery, nor is it likely to do so . . . . It is an operation not within the range of practical surgery." In expressing this opinion Mr. Page has no doubt been influenced by the difficulties and dangers before referred to, but I would submit that they have no real existence.

Mr. Erichsen, in the last edition of his 'Surgery,' says, "The operation is not necessarily dangerous, it does not appear often to have hastened death, and has certainly in some cases afforded relief." With regard to the latter point I will discuss that when reviewing the cases I have succeeded in collecting, and I think a description of the mode of operating that I have adopted will show more briefly than anything else the way in which many of the so-called objections to the operation may be removed. In the first place the operation has been generally objected to on account of:

order to which the surgeon must lay bare the fractured vertebræ with a scalpel and replace or else remove such fragments as injured the spinal marrow."

1. Hæmorrhage.
2. Difficulty in clearing the neural canal.
3. Physical difficulties of treating the fractured vertebræ.
4. The hopeless nature of the damage of the spinal cord.
5. Septic infection.

Of these objections we may with advantage consider Nos. 1, 2, and 3 together. In the course of some experiments upon the spinal cord, which are fully detailed in 'Brain,' vol. ix, 1886, I found that the mode of operating upon the spine for complete exposure of the bones as given in the text-books, namely, by removing the muscles from the bones by means of a blunt instrument, so far from being the best means for preventing hæmorrhage, is the easiest way of producing it. The knife must be freely and rapidly used while the soft parts are strongly retracted. It must be noted here that in every case I have found it necessary to divide the deep fascia, not only along the spinous processes, but also at right angles opposite the middle of the incision in order to prevent it resisting proper separation of the sides of the wound, indeed, it may in some extreme cases be found necessary to divide the vertebral aponeurosis at more places than one. It need hardly be added that this division of the fascia has no influence upon the rapid healing of the wound. The free bleeding which follows the separation of the muscles from the bone is best met by seizing what bleeding points can be seen with Wells's forceps and then tightly and quickly packing the incision on one side of the spine with dry sponges while the operation is proceeded with elsewhere. In this way very free oozing may be arrested in a few minutes, and the time spent in waiting for it to stop is certainly not lost, because the subsequent division of the bones can only be properly carried out when the wound is perfectly dry, as it is when the above-mentioned method has been followed. The periosteum is best reflected, without impairing its vitality, by scraping the bones with a suitably curved elevator after the mass of muscle has been turned aside.

Next with regard to the removal of the laminæ of the



vertebræ. If, of course, there is a fracture of the laminæ or of the spine it will be detected at once by seizing the bones individually in strong forceps and shaking them (Erichsen), the fragment being easily extracted by dividing with a knife the ligamentous attachment, and no damage will be done to any important part if the edge be directed towards the bone. If now, however, the spine be perfectly uninjured, as in the foregoing case, it becomes a matter of great interest as to how we may most quickly remove the bones. From numerous experiments on dogs I have adopted the following method: The spinous processes of the vertebræ whose laminæ are to be removed are cut through close to their base by very powerful bone forceps. This is readily done in a few seconds and we then have the laminæ forming a continuous if irregular plate, and this can be perforated with a trephine with the usual precautions. The trephine should be almost as large as the diameter of the neural canal, this of course varying with the region operated on, the age of the patient, &c. If more than one arch is to be removed it will be better, by means of an angular saw, to partly cut through the laminæ along the lines of the sides of the neural canal, and then the division of the bones can be completed with a bone forceps.

As in all these cases the wound cavity is necessarily deep, its walls steep, and relatively very unyielding, I have devised a form of bone forceps suitable for this stage of the operation. They simply consist of two ordinary bone forceps cutting blades set at an angle of about  $120^{\circ}$  to two short arms, which meet at the hinge and which are continuous with the ordinary long handles, the whole being bent at the hinge in a sharp curve, so that they can be employed to cut horizontally at the bottom of the cavity. More difficult than the incision of the bone is the removal of the ligamenta subflava. These can only be quickly and safely got rid of by steadily cutting with a sharp knife. After removal of the bone, as is well known, we find the dura mater covered with very vascular fat of a peculiar nature. This fat and loose connective tissue, if not treated

in the way about to be described, may be very troublesome indeed in causing free oozing of blood, and at the same time owing to its elasticity in obscuring the proper view of the dura mater. The numerous vessels supplying it of course come from the spinal arteries and the vertebral plexus of veins. Consequently, these are best avoided by keeping the incision in the fat strictly to the middle line. When this is done there will be very little bleeding at all, but at the same time the dura mater is completely covered by the fat. It can, however, be practically completely got rid of from the field of operation if it be retracted with broad retractors, and pressed against the sides of the neural canal for a few minutes or seconds while the dura mater is opened. This fatty tissue being very spongy seems to shrink under the pressure and remains practically out of sight during the remainder of the operation.

The next point to be considered is the treatment of the dura mater, &c. The dura mater, if opened in the middle line, will be found to admit of quite sufficient retraction to either side to expose the whole spinal cord and the subdural space. If of course the longitudinal incision in it be very short, say less than half an inch, it will be necessary to make a transverse incision as well in order to expose the whole breadth of the subdural region. But, as in the present instance, if incision be at all long, it is quite sufficient to restrict it to the middle line.

The next practical point is the escape of the cerebral fluid since on the first opening of the dura mater the cerebro-spinal fluid wells up very freely indeed, fills the wound and prevents anything like accurate handling of the spinal cord. The best course to pursue is to keep mopping it out of the wound cavity with a sponge so long as it flows. If the patient be not moved and if the spine be horizontal and the head not raised, the flow of fluid will soon cease, and the spinal cord be then very freely visible. After inspection the spinal cord should be examined very gently by palpation, it being pressed against the bodies of the vertebræ in front, so as to reveal any change in its density. If

it be suspected that some fragments of bone or a new growth may be pressing against the anterior surface of the cord from one of the vertebræ, it can best be detected by carefully passing an aneurism needle around the side of the cord, and exploring this aspect of it. In mentioning of course the opening of the dura mater, the escape of fluid, &c., due care will be taken to notice in the first place whether the dura mater is of normal appearance and whether there is any indication of its being inflamed or distended. It need hardly be stated here that of course if there is hæmorrhage beneath it it will appear dark, if pus yellow, &c. In cases where the theca is pressed backwards against the laminal arches, either from old traumatism or caries, &c., great care must be taken in perforating the laminæ with the trephine, but still more in raising the bone from the theca, for in such cases the dura is adherent to the anterior surface of the laminæ by firm fibrous adhesions, these requiring division with the knife.

A more difficult question, and one which requires experimental investigation at the present time, is the problem under what circumstances it is advisable to suture the incision in the dura mater or to leave it open. In the foregoing case, although the incision was relatively of very great extent, it was left open, but there can be little doubt that the union of the wound at the bottom of the uppermost drainage-tube canal would have been much more rapid if the dura mater had been even imperfectly closed by fine sutures.<sup>1</sup> There is another point which seems to me to be of practical interest, even if its existence be only theoretical. This is the possible cicatricial adhesion of the floor of the wound to the posterior surface of the dura and cord, and to the posterior roots of the nerves entering the same.

It has been long known to physiologists since the researches of Schiff in 1851, that the posterior columns of the cord are conductors of painful impressions, and this can be demonstrated on an animal which is completely

<sup>1</sup> I have since sutured it with success.

narcotised with ether, so as to prevent the appreciation of pain, in the following manner. If in such an animal the various columns of the spinal cord be successively touched with a sharp point or other mechanical irritant, no reflex, *i. e.* involuntary, movements will result, except when the posterior columns are touched. This fact, as well as another mentioned below, is worthy of notice, inasmuch as it makes a high degree of narcotisation necessary for the performance of the operation under the best possible conditions. The other fact I now refer to is the sensibility of the dura mater. It is not apparently generally known that the spinal dura mater is an exceedingly sensitive membrane. In the dog this is particularly noticeable, and even in an animal perfectly narcotised with ether, reflex movements will occasionally occur when this membrane, like the posterior columns of the spinal cord, is mechanically irritated. It is very necessary, therefore, that when the dura mater is about to be seized in forceps with the view of opening it, or when similarly any delicate incision is to be made in it or into the neighbourhood of the posterior column of the cord, that the patient should be very deeply under the influence of the anæsthetic to prevent any unconscious reflex start, which might lead to very unfortunate results.

To return, it will be readily understood now that possibly as before stated the wide cicatrisation together of the floor of the wound, the dura mater and the posterior roots of the nerves might produce adhesion which would cause pain if the spine were freely moved. This, however, after all may be purely imaginary since in the present case, where all these conditions must exist, the pain which occurred after the operation was not attributable to this condition, since in the first place it was merely a continuation of that which the patient endured before the operation and moreover has now (Jan., 1888) practically disappeared.

4. *The hopeless nature of the damage of the spinal cord.*

The consideration of this part of the subject is important

of course only in those instances where the cord is diagnosed to be completely softened or where the operation is undertaken for the purpose of relieving the results of fracture of the spine, the whole bearing of which we may very properly now consider. I am the more anxious to do so since I can make my meaning clearer by referring to a case of Dr. Buzzard's, in which I performed the operation last summer. The patient had fallen down a quarry and was sent up from Derbyshire to the National Hospital for the Paralysed and Epileptic in a very critical condition. There was absolute paraplegia as regards movement and sensation together with complete loss of control over the bladder and rectum. The urine was already alkaline and contained muco-pus. But the worst feature in the case was the existence of very severe and acute decubitus. Thus there was a large spreading sore over the whole breadth of the sacrum, extending especially deeply on the left side, sores over both heels, and a bleb on the left thigh.

It was very clear indeed to Dr. Buzzard and myself that unless the man was relieved from the most urgent symptoms he must speedily die. Examination of the spine showed that the spinous process of the eleventh dorsal vertebra was apparently broader than natural and a little more prominent, and at the same time very distinctly tender, in addition to which the patient very distinctly referred to this region as being the source of his weakness.

It was therefore decided to explore the seat of the fracture and if possible to remove any portion of the bone which might be pressing upon the spinal cord. The patient being placed in a prone position, and anæsthetised with chloroform, a longitudinal incision was made over the prominent vertebra. The soft parts, as before detailed, reflected, and then on grasping the spine of the eleventh dorsal vertebra it was found to be movable, but jammed forwards between the vertebræ above and below it. It was therefore seized in lion forceps and removed by cutting all the ligamentous bands attached to it. When it was extracted it was evident that the posterior surface of the dura-matral sheath

was pressed backwards against the laminæ of the tenth vertebra. This therefore was also removed. The dura mater appeared perfectly normal. The theca therefore was not opened posteriorly, but on exploring its anterior surface and the bodies of the vertebræ with an aneurism needle a small puncture was made into it from which there escaped perfectly normal cerebro-spinal fluid. There was no evidence of any previous severe compression of the dura mater or its contents, and no evidence of hæmorrhage into the same. The wound was therefore closed and a drainage-tube placed opposite its middle; it was dressed strictly antiseptically. The further progress of the case was one of much interest; although the operation made no difference whatever in the motor paralysis and only slightly improved the sensory paralysis, it completely arrested the acute decubitus, the sores ultimately healing firmly, and what is still more interesting, from the time of the operation the urine became acid.

The drainage-tube was removed on the second day and the wound was completely healed at the end of seven days without a trace of suppuration.

In this case no doubt the spinal cord was momentarily jammed at the time of the accident so severely as to practically, *i. e.* functionally divide it. Though laminæ were found compressing it at the time of operation, nevertheless they did not do so so severely as to thereby alone cause the excessive degree of the symptoms, therefore it would seem that the cord, as suggested, must have been compressed at the time of the accident. Now, this is just a case in which if any attention had been paid to the ruling before quoted, the patient's life would have been lost, and indeed it must be obvious that, considering the necessarily small amount of information on this subject, it should be our duty to operate in every case, since we may possibly do some good, and certainly, if proper antiseptic precautions be taken, we can, to use Mr. Erichsen's words, do no harm. This question of the damage to the spinal cord not appearing to discount the possible benefit of surgical interference so much as has been

hitherto expected, gains additional illustration from the case which forms the subject of this paper, since nothing could well have appeared more hopeless than the indentation of the spinal cord produced by the tumour. This indentation appeared to divide the lateral column completely, and yet, owing doubtless to the gradual character of the compression, the restoration of motor and sensory function has been complete. I would repeat therefore that, so far from its being unjustifiable to operate on the spine owing to the possibility of the cord being hopelessly damaged, it seems to me to be criminal not to operate.<sup>1</sup>

##### 5. *Septic infection.*

The possibility of septic injection following this operative procedure is of course exactly the same as that which attends any surgical interference, and needs only to be guarded against in precisely the same way, namely, by the Listerian principles of antiseptic surgery. No special liability to septic infection attends wounds in the region of the vertebræ, though of course the subdural space is undoubtedly an unusually favorable nidus for the organisms of putrefaction. As, however, I have repeatedly urged in discussing the surgery of the cranial cavity, this danger can be removed by the use of powerful disinfectants in a strong solution, *e. g.* 5 per cent. carbolic acid solution, &c., by irrigation of the wound during the operation, and this irrigation is afforded with the least inconvenience by the spray. The drainage of the wound is so extremely easy since the patient usually lies supine, that it can be completely provided for by a drainage-tube kept in for not more than forty-eight hours.

Summing up therefore, I think I have shown reason for regarding the operation of trephining the spine as a comparatively easy one, safe and justifiable, and that its reputed dangers are no more than those incidental to all wounds,

<sup>1</sup> Since this was written I have trephined the spine in two more instances, in both the wound healing without any complication whatever; one, a fracture: the other a case of complete paralysis of all four limbs, &c., from severe caries of the second and third cervical vertebræ is now rapidly regaining power, being able to move both legs on the eighth day after operation, and the upper limbs later.



the only peculiarity being the fact that septic meningitis is practically a fatal accident, the avoidance of which, however, is well understood and provided for in 999 cases out of 1000.

We will therefore pass on to the review of the similar cases collected in the accompanying table.

*Tumours of the membranes of the spinal cord.*—I have been able, by referring to text-books and journals, to find accounts of fifty-eight cases, which I have arranged in the following table, which does not pretend to be exhaustive but I hope is sufficiently illustrative of the subject to warrant the conclusions which terminate this communication.

In this Table the facts relative to each case, so far as they have been recorded, I have arranged under headings which seemed to indicate the salient points upon which our future information must necessarily be full to admit of our making a correct diagnosis. Unfortunately, as will be seen, even the most fully recorded cases do not admit of close contrast one with another, owing in the vast majority of instances to the fact that their real nature was wholly unforeseen. Hence it may be well at some future time to draw attention to one or two points in comparative diagnosis, which need a more complete elucidation than is apparently forthcoming from the clinical records at our disposal. Especially is this required for cases where the active source of pressure is a parasitic cyst, and which, therefore, can only be treated by surgical operation. A collection of such cases has recently been made by Dr. Maguire, to whose paper in 'Brain,' January, 1888, p. 451, reference must be made for details, but I have inserted a few cases in the table by way of comparison.

The special want of information will be seen at once in the summary of the facts detailed in the Table, and in the history of the case, the subject of this paper.

The summary of the facts in the Table will be best arranged in the order of the headings to each column as follows :



*Column 2. a. Age.*

Among the preliminary facts, the age of the patient, the subject of any of the diseases included in the table, is of course an important matter, since it will give aid sometimes in the diagnosis of the nature of the growth.

**A.—EXTRADURAL GROWTHS.**

1. *Lipoma*.—It is interesting to note that in the four cases of lipoma the extremes of the ages were 10 months and 4 years respectively, the average being  $2\frac{1}{2}$  years. Since the nearest approach to this age is to be found under the heading of intradural tubercle (see below), it is evident that if we have presented to us a patient of such an early age with symptoms of compression of the spinal cord, the assumption that the growth is lipoma will be very strong indeed. For further discussion of this important point see 'Nature of the Growth,' Column 24.

2. *Sarcoma*.—The kind of tumour which occurs next in the order of age is sarcoma, the average age of the patients being 18 years.

3. *Echinococcus*.—Next to this sarcoma is echinococcus (of course almost invariably extradural in any case), which attacks patients of an average age of 34 years (possibly less).

4. *Tubercle*.—It is interesting to find that tubercular mischief outside the spine causing fatal compression of the cord, apparently occurs in patients of an average age of 39 years. I say apparently because the fatal cases are not numerous, whereas of course we frequently see non-fatal instances of pressure of the cord from extradural tubercular disease, usually spinal caries, in children.

The foregoing are the commonest forms of extradural disease. The rarer, such as scirrhous or myxoma, occur beyond middle life, at 48 and 53 years in the table, as of course would be anticipated.

## B. INTRADURAL GROWTHS.

1. *Myxoma*.—Included under this heading are pure myxoma, myxomata in which there is a considerable amount of fibrous tissue, and which therefore might by some be considered worthy to be put into a separate class entitled *Myxofibroma*, and lastly cases<sup>1</sup> in which no diagnosis is given, but which, nevertheless, from the description, are evidently of a similar nature. Under any circumstances the cases of undoubted myxoma outnumber the instances of any other kind of intradural growth. In a total of eleven cases the extremes of ages were 19 and 60 respectively, the resultant average being just over 43 years of age.

2. *Fibroma*.—At almost exactly the same age as myxoma, fibroma occurs; thus the average of the six cases was 44 years.

3. *Sarcoma*.—Similarly, sarcoma is noted to occur at the average age of 41 years.

4. *Psammoma*.—Contrasted with the foregoing tumours of youth and middle life on the average is psammoma. This growth, which has been recognised for many years as epitheliomatous, it is instructive to see occurs at an average age of 51 years. This is in harmony with the general facts recorded of the ages at which any of these neoplasms are likely to arise.

5. *Tubercle*.—One disease alone remains for special notice, namely, tubercle, which in its intradural form was found present on an average at the age of 18·5 years. Reference to the extradural form will show that that occurred most commonly at 39 years, due reservation being made as already stated. For general purposes, however, we may conclude that if we have evidence of an intradural growth in a person beyond 30 years of age, it is almost certainly not tubercle.

<sup>1</sup> As will be pointed out later, it seems likely that the results of traumatic hæmorrhage are included in this class.

*Column 2. b. Sex.*

Of the total number of fifty-seven cases the sex is recorded in fifty-four, of which twenty-four were males and thirty females.

The preponderance of the female sex is the attribute of the intradural growths as contrasted with the extradural. Thus, in the former class of thirty-six cases fourteen were male and twenty two female, while of the latter class of eighteen cases ten were male and eight female.

Why the female sex should more especially suffer is not easy to see. If the views expressed further respecting traumatism are correct, then the difficulty will be in a measure removed, for in the course of parturition we have an obvious source of intraspinal traumatic lesion.

This subject, however interesting, scarcely in our present knowledge admits of further profitable discussion; we will therefore pass on to consider the next point, viz. the alleged cause of the disease in each case.

*Column 3. Alleged Cause.*

The discovery of a cause of the appearance of a new growth is in most cases a matter of considerable difficulty, and the origin of intraspinal mischief especially. It will, however, appear that some forms of new growth, either without or within the theca, do in their minute structure clearly suggest their mode of origin and source.

**A. EXTRADURAL GROWTHS.**

1. *Lipoma*.—In three of the four cases in the Table the fourth case probably supplies the clue, viz. congenital abnormality. The fat, which normally occupies much of the space between the theca and the neural canal, is highly vascular, and can easily be understood to occasionally take on a hypertrophic condition, which would lead to the fatal issue.

2. *Sarcoma*.—In three of the five cases to which a cause is assigned, we find it asserted to have been respectively “fall on the back,” “exposure to cold,” and secondary to primary disease elsewhere. I will return to the two former of these in summing up.

The causation of the remaining diseases does not call for special notice, owing to their pathology being known, with the exception of fibro-chondro-lipoma, in which again exposure to cold is alleged as the probable cause of the origin of the tumour.

### B. INTRADURAL GROWTHS.

1. *Myxoma*.—Of the eleven instances of this tumour, cause is assigned in seven cases as follows :

Traumatism . . . . .	2 cases.
Exposure to cold . . . . .	2 cases.
Parturition . . . . .	1 case.
Suppression of menses . . . . .	1 case.
Fright . . . . .	1 case.

Possibly we should associate the third alleged cause with the first, and the fourth with the second.

2. *Fibroma*.—Of seven cases in which cause is alleged it is described as follows :

Exposure to cold . . . . .	3 cases.
Traumatism . . . . .	2 cases.
Chlorosis . . . . .	1 case.
Fatigue . . . . .	1 case.

3. *Sarcoma*.—For this growth we find :

Exposure to cold . . . . .	2 cases.
Traumatism . . . . .	1 case.
Fit of anger . . . . .	1 case.

4. *Psammoma*.—Cause given only once. Stated to have come on soon after parturition. ? Traumatism.

The other intradural conditions, such as tubercle, parasites, &c., require no description.

On addition of all the alleged causes of both extra- and intradural growths we find them to stand as follows :

Exposure to cold . . . . .	8 cases.
Traumatism . . . . .	8 cases.
Mental shock . . . . .	2 cases.
Chlorosis . . . . .	1 case.
Fatigue . . . . .	1 case.
Congenital . . . . .	1 ? 4.

Considering that these alleged causes are given for only thirty-five<sup>1</sup> instances of disease, it is very remarkable that in almost one half of the total number of the cases thus recorded the cause should either be attributed to traumatism or exposure to cold, and this fact is of course greatly strengthened by consideration of the whole number of twenty-one alleged causes no less than sixteen are ascribed to these excitants. To this must be added certain facts from the histories of the cases, which are but briefly alluded to in the Tables. Thus, in several instances of the traumatism and in many instances of exposure to cold, the leading and earliest symptom, *i. e.* pain, was noted to commence directly after the sufferance of the alleged cause. But more than that. We have, I think, direct evidence on this point of a more valuable kind, *viz.* that afforded by anatomical investigation, whilst if from the table we extract the eight cases of traumatic origin, we shall find that three must be put aside because the tumour was solid, or because not sufficient facts were known about it to enable us to express an opinion bearing upon the question at issue. Of the remaining five, in two the symptoms were observed to follow directly upon the injury, so there is no question as to the real relation existing in them between the supposed cause and effect. These two cases are Nos. 24 and 44, and though classified according to the diagnosis as myxoma and fibroma respectively they are indubitably cystic formations so commonly set up by traumatic hæmorrhages in the membranes of the central nervous system. The records make this point perfectly clear, especially in Case 44, where the altered blood pigment is clearly described.

<sup>1</sup> The origin of the remaining twenty-three being known, *e. g.* tubercle, &c.

Passing now to Cases 46 and 32. In each I have had the opportunity of microscopically investigating the growth very thoroughly, and in each I have found scattered through the whole growth numerous collections of hæmatoidin granules, in many cases enclosed in corpuscles, as is so frequently seen in the neighbourhood of old extravasations of blood. At the same time there were no evidences whatever of recent hæmorrhages into the tumour substance such as occur in myxomata. Case 28, also classified as myxoma from its remarkable identity with 32, I strongly suspect to be due to the same morbid process, and this notion gains weight from the history of the case.

### HISTORY.

#### *Column 4. Personal.*

The personal history in such a heterogeneous mass of cases has little value. It is only of somewhat painful interest to note that in almost all instances of these invariable fatal cases, the patient was in perfect health before the commencement of the symptoms and nevertheless was condemned to an extremely painful and lingering death, although as a rule the mischief could have been readily removed by surgical interference.

#### *Column 5. Family.*

In the same way there are no facts of noteworthy value to be drawn from the records of the family history.

#### *Column 6. Total duration of Symptoms.*

##### *Extradural growth—*

Lipoma . . .	Average about	1½ years.
Sarcoma . . .	„	9 months.
Echinococcus . . .	„	6 months.
Tubercle . . .	„	1½ years.
Scirrhus . . .	„	1½ years.
		<hr/>
		5 yrs. 7 mos.

General average about 1 year, 1 month.

*Intradural growth—*

Myxoma . . .	Average 4½ years.
Fibroma . . .	„ 3½ years.
Sarcoma . . .	„ 2 years.
Psammoma . . .	„ 3½ years
Tubercle . . .	„ 8 months
Parasitic cysts . . .	„ (?) 9 months
<hr/>	
14 yrs. 6 mos.	

General average about 2 years 5 months.

From these figures it is evident how much more chronic are the symptoms when the mischief is within the theca than when outside it. Doubtless this is simply owing to the prevalence of the simpler forms of neoplasm among those growing from the spinal membranes. To whatever cause it may be due the fact is important in assisting the probable diagnosis.

*Column 7. Course of Symptoms.*

Nothing obviously is of more importance than the special manner in which the symptoms are gradually developed. Unlike those occasional instances in which a cerebral tumour suddenly produces pressure symptoms, the spinal cord tumour seems practically never<sup>1</sup> to initiate such a condition. The only noteworthy exceptions being for the most part parasitic cysts which sometimes owing to the hydrostatic conditions under which they exist, suddenly enter the neural canal in some position of flexion of the spine. See Case 16.

Review of the whole number of cases, regarding them all as simple instances of mechanical pressure on the cord, is perfectly justifiable for the correct estimation of the relative sequence of a few symptoms, the remainder being considered in their development under their special headings in Columns 8 to 20.

<sup>1</sup> Sir B. Brodie in the 'Lancet,' pp. 378, 379, gives a case in which an intramedullary growth produced sudden paraplegia.

*Column 8. I. Extradural Growths.*

Of seventeen cases belonging to this class in which the course and development of the symptoms was fairly recorded, the first symptom was pain in seven cases, motor paralysis in six, the two combined in one case, motor and sensory paralysis combined in another, and finally muscular spasm in one case. Most frequently in the subsequent development of the chain of symptoms, sensory paralysis came later than motor, frequently the patient noticed the two to be developed simultaneously. Altogether twitchings of the muscles were recorded historically in but three cases see, however, Column 12.

The symptoms as a rule indicated diffuse pressure within the neural canal, for in only five<sup>1</sup> was there an approach to transfer of symptoms from one side to the other (right to left in three cases left to right in two). It is proper to add, however, that in four other cases the symptoms showed a tendency to be most marked on one side (the right in four cases the left in one).

Summing up we have thus ten cases in the seventeen in which there was an incomplete unilateral character in the symptoms.

However, the most important point in the development of the symptoms is of course the march, and in this extradural division of the cases it is clear that the commonest march would be **Pain, Motor paralysis, Sensory paralysis**. It being very distinctly understood that it is common in this condition to have the motor and sensory paralyses in close combination both in degree, relatively speaking, of course, and in time.

*II. Intradural Growths.*

In contrast to the comparatively frequent occurrence of ill-defined arrangement of symptoms in the extradural growth,

<sup>1</sup> i. e. 29 per cent.



we have in the intradural a most remarkably constant march. Thus from a total of thirty-three available cases twenty-one begin within pain as the first symptom, four pain combined with motor paralysis as a first symptom, and but six with motor paralysis alone. The subsequent march is just as striking, the loss of motor power preceding sensory paralysis in the very large majority of cases, indeed only in four instances out of the total of thirty-three did sensory paralysis precede motor. Therefore the march is easy to write in these intradural cases, being **Pain, Motor paralysis, Sensory paralysis.**

Of greater interest, and for diagnostic purposes of greater value, are the facts relating to the transference of the symptoms from one side of the body to the other, the possibility of localisation of the growth being thereby of course greatly increased.

Careful observation of such cases (as, for example, No. 50) indeed reveals, in some instances, pathological effects such as make the condition tantamount to hemisection of the cord, so that we have reproduced clinically the classical experiment of Brown-Séquard. But in the majority of cases we have to derive our information from the unskilled observation of the patient or the friends, so that at the most we learn that transference did occur. The transference in this division of the cases is very different to that in the extradural, inasmuch as in this variety the interval between the invasion of the limbs successively affected is usually weeks and occasionally months.

Of the thirty-three cases such marked transference was noted in sixteen cases, *i. e.* 48 per cent., which is a great advance upon the 29 per cent. of the extradural growths. Of these sixteen cases in nine instances the transfer was from the left to the right, in seven from right to left.

Grouping both divisions of the case together we find that as soon as the paralytic symptoms are well developed, the pain (for fuller details of which see the discussion of the next column) becomes horribly exaggerated, then decubitus occurs, and later the patient dies pyæmic or uræmic, by

septic absorption from the bedsores or from the decomposition of the urine.

*Column 9. Pain.*

From the time of Cruveilhier the pain accompanying the development of intraspinal tumours has been reckoned with paralysis to be the most noteworthy symptom in the affection. Its course is almost always insidious, excessively rarely is it sudden in its onset, being then obviously owing to hæmorrhage or rupture of a cyst. At the first and indeed throughout the course of these cases they have almost invariably been diagnosed as rheumatism; consequently it behoves us to examine closely into its character to see whether there are not features peculiar to it which should prevent such a melancholy mistake in the future. We will therefore go through the records of the pain in each case from this standpoint.

First we note the immensely important fact that however severe the pain may be it is never referred to any part above the seat of the lesion. To this there seems no exception. Besides Case 32 there are several which show this most strikingly. While thus the symptom is never placed above the lesion it is only localised below the lesion, a matter of extreme import to the operator in search of a tumour, &c.

If possible it would be most interesting to determine exactly the localisation of the pain as contrasted with the actual position of the tumour, because we should thus have, owing to the constancy of pain as a symptom, a powerful addition to the array of diagnostic guides. Although the records at our disposal are unfortunately greatly wanting in this respect, those cases which do point to some conclusion on this question had better now be referred to in a little detail.

Case 7. In this case the growth was situate from the sixth to the eighth dorsal vertebræ, *i. e.* bodies. The pain was referred to the distribution of the ninth dorsal nerve. Simi-

larly in Case 32 the growth was opposite the third dorsal vertebra (body), the pain being distributed in the area supplied by the fifth nerve. In these two instances the difference between the position of the growth and the localisation of the pain is clearly due to the anatomical relations of the nerve-organs and roots to the vertebræ, and something more, viz. the as yet (in the human being) imperfectly known course of the fibres in the spinal cord. Thus in Case 32, as is stated in the history of the operation &c., the growth was diagnosed to be opposite the highest root of the fifth nerve, *i. e.* localising it by means of the pain and anesthesia, but as a matter of fact the tumour was situated four inches above the zone of anæsthesia. It is possible that the actual pressure of the growth upon the cord produces much less effect on the intramedullary fibres than one would expect, and this would seem to explain why the ascent of the distribution of anæsthesia to the level of the lesion is so very long in completely developing. Extreme cases are those in which, as in Cases 11 and 13, extradural pressure, the lowest point of which was the third dorsal vertebra in the one case and the fourth dorsal vertebra in the other, produce pain referred to the lower limbs. The other extreme, viz. perfect localisation by means of the nerve, is to be seen in Case 28, where invasion of the fifth dorsal root upon which the tumour was sessile caused severe pain in the corresponding intercostal space. Probably also Case 46 is of a like character, an example of intermediate cases for which no explanation appears forthcoming save such as I have suggested in No. 41, where we have the pain for a long while referred to the distribution of the ulnar nerve most particularly, although the growth did not descend lower than the fifth cervical vertebra, the ulnar nerve of course deriving most of its fibres from the eighth cervical and the first dorsal. In this last case there were certain vague pains felt in the neck and shoulder, which of course might certainly be taken as suggesting possible affection of the cervical nerves as high as the fifth, but these were evidently insignificant compared

to the localisation of the pain to the distribution of the ulnar nerve. The only conclusions then that we can draw to guide us in this matter are, firstly, that if the pain be extremely well defined in the course of one nerve of one side then a lesion may be diagnosed to exist in the course of that nerve and so it may be localised with ease; secondly, that where there is no delimitation of the pain, but where the anæsthesia is apparently well marked and in accord with nerve-supply, it is necessary to be most cautious and to define if possible the upper border of the hyper- or paræsthetic zone which so usually surrounds the upper limit of the anæsthesia, but applying what has gone before, the proper course to follow will obviously be the exploration of the neural canal and its contents at the highest point that is suggested by any definite symptoms, and among such we should include even slight paræsthesia. Pain of a constant dull aching character seated in one fixed point in the spinal column is of course most valuable as indicating the focus of disease, but caution is requisite here again, and unless the limitation of the pain is very distinct it would be best to regard it as of slight value. If, though slight, it is accompanied by a symptom of which very little notice is taken, to judge by records, but one nevertheless of great value, namely, a sensation of weakness felt by the patient at that point in the spinal column, and if this sensation, which is of course subjective, is heightened by fatigue, then it assumes a leading position among the localising symptoms.

We may now proceed to consider<sup>1</sup> the character of the pain with regard to its diagnostic value. Noting unusual forms first we must begin with the subjective sensation of burning, which occurred as frequently as in five out of the fifteen cases in which the pain was carefully described. In Case 32 this was extremely marked, the patient complaining that the whole of the left side and lower limb appeared to be red-hot and burning. In another patient the pain was described as roasting. This consequently

<sup>1</sup> In the intradural division only.

would appear to be an important fact to remember in dealing with painful conditions of the spine. The most frequent form of pain is that common to many affections, namely, shooting. In two instances it is described as pricking, in one as gnawing, and in one as stabbing, but little value attaches to solitary statements of this kind, especially as a full description is so seldom given. The next point is the relative amount of pain excited by the presence of the tumour. This, of course, is only to be correctly estimated in the intradural division of cases. Careful examination of the cases in which the pain is noted shows that even after eliminating possible errors it is a more prominent feature in those cases where the pressure is directed postero-anteriorly, and antero-posteriorly, than in those in which it is directed laterally. This is easy to understand in view of the fact that in the first two directions the pressure will directly influence the extremely sensitive posterior columns. The exceptions to this generalisation may be very striking, as, for instance, in Case 32.

It will not escape notice in the tables how almost invariably the pain in the lower limbs ascends from the soles of the feet in precisely the same manner as the anæsthesia is almost always subsequently developed.

Considering the extreme frequency of pain from tumours pressing on the spinal cord it is worth while glancing at those few instances in which pain is not recorded. In none of the cases of extradural lipoma is pain recorded. Probably this omission may be attributed in great part to the age of the patient, and has consequently been omitted from the records, but again it is not recorded in Cases 33, 35, 49, 57. Unfortunately these latter are just cases in which the notes are not so complete as could be wished, so that it would be hazardous to guess what the absence of record signifies. In view of the general obscurity of the subject, however, it is worth while to draw attention to the point.

*Column 10. a. Paralysis of Motion.*

Few things are more regrettable than the almost complete absence of record as to the mode in which the motor paralysis in these cases successively invades the various segments of the limb. So far as can be made out the advance of the paralysis is from above down. The patient in five cases of paralysis of the lower extremities was unable to stand before loss of the movements of the leg when lying down, and this form of early paresis was not necessarily accompanied by such sensory paralysis as produced ataxia. In one case of paralysis of the upper limb the result was different, the fingers being stated to be paralysed while the shoulder was only weakened. It is most desirable that this subject should be elucidated as soon as possible for the sake of prognosis as well as diagnosis, and all observations should be complete for each segment from the hip to the great toe, the representation of the latter being specially interesting.

*b. Paralysis of Sensation.*

The gradual abolition of sensibility appears in all cases to advance up the limbs from the soles of the feet. Here again the difference between the appreciation of various forms of sensory stimulation needs much careful examination in the future, and notably the contrast between pain, touch, and temperature (see Case 28).

*Column 11. Reflexes.*

In every case, without exception, if the cord were pressed upon, the reflexes both superficial and deep became greatly exaggerated. As the cord became completely destroyed

and descending degeneration and wasting marked, so the reflexes were gradually lost, the abolition beginning almost invariably with the plantar and passing upwards.

*Column 12. Spasms with Clonus.*

According to well-known facts, spasms and clonus ankle (and knee ?) were noted in the large majority (61 per cent.) of the intradural cases and in 35 per cent. of the extradural. It is noteworthy that among the latter they were only recorded as accompanying the development of lipoma and echinococcus cysts. The reason of this is not apparent.

The genesis of such spasms is slightly illustrated in the Table by but one case, No. 28, in which the spasms are noted as occurring on the same side as the tumour. Hence we may obtain, on further examination of future cases, evidence as to the direct effect of pressure in this connection.

It is further worth mentioning that in one case in which the spasms were originally most severe they disappeared one month before death, *i. e.* when the cord became so disintegrated as no longer to react to the irritation.

*Column 13. Rigidity.*

Closely connected with the foregoing column is that of rigidity. Like spasm and clonus this is very much more frequent with intradural than extradural growths, the percentages of recorded occurrence being respectively 50 per cent. and 15 per cent. Consequently, there is good reason for supposing that the existence of marked rigidity is evidence of intradural mischief.

A point of interest now arises. In fourteen cases of intradural growth in which it was recorded, in eleven the attitude assumed by the contracted limb was that of flexion, in three that of extension. One case suggests a

cause of this difference. In one instance in which the tumour was found on the right side of the spinal cord the right lower limbs were the seat of spasm in flexion, whereas the left lower limb was in extension. It would seem as if the growth specially induced irritation effects according to the intensity of the pressure employed. Thus, in the initial stages of the "pressure myelitis" the spasms may be those of extension, while in the final condition flexion predominates. The same thing may be noticed in the pressure myelitis resulting from fracture, dislocation of the spine, &c.

*Column 14. Decubitus.*

The formation of bedsores is of course the rule, and these are noted almost invariably in the cases of intradural growths as being terribly extensive. In many instances the buttocks are deeply excavated as far as the trochanters. In two cases (Nos. 47 and 32) the bedsore was situated on the same side of the body as the tumour, just as cerebral decubitus is seen on the opposite side to the nerve lesion.

For the effects of the decubitus see p. .

*Column 15. Nutrition.*

The nutrition of the paralysed parts is variously described as affected. Thus, in a few instances the change is mentioned as local, twice in the extradural and five times in the intradural growths. There is of course obvious wasting when the growth penetrates an intervertebral foramen and destroys a nerve-root, but the instances in which local wasting occurs, *e. g.* of one lower limb as in Cases Nos. 24, 41, when the pressure is general are very rare. In eleven cases of both kinds of growth the wasting is described as general, and it may be that this general emaciation has led to supposed observation of local atrophy.

As a rule the nutrition is not impaired unless the paralysis be complicated with pyæmia, &c.



*Column 16. The Spine and its Physical Examination.*

This is a very interesting point, but not one from which much can be learnt, for out of the thirty-one cases (in both groups) in which the examination was made, it is distinctly stated in thirteen that nothing could be discovered.

Putting aside the six cases (five extra- and one intradural) in which the problem of diagnosis was obviously solved by the presence of an external tumour solid or containing fluid, we may proceed to note some one or two important facts, although the number of cases remaining at our disposal is only twelve.

A review of these shows that the alterations in the spine produced by an *intradural* growth are as follows :

1. Tenderness on percussion.
2. Stiffness and weakness.
3. Curvature usually lateral.

The relation of each of these to the seat of the tumour is the most valuable evidence they offer, and that is as follows.

Perhaps the correspondence or want of the same will best be shown in parallel columns. Thus :

Tenderness on pressure was felt at the	The tumour was situated at
Mid-dorsal region.	5th D. nerve.
10 D. V. and downward.	7 to 10 D. V.
1 to 4 C. V.	4 to 5 C. V.
5 to 7 C. V.	4 to 5 C. V.
1, 2, 3, 10 D. V.	2 to 3 D. Nerve.
7 C. V.	1 ? and 5 D. ?
6 D. V. (spinous process).	3 to 4 D. Nerve.

From this Table it appears that practically without exception a tumour occurring in the dorsal region is higher than the spot described as tender on pressure. In the cervical region this generalisation does not seem to hold so closely. It need hardly be pointed out that the inclination of the nerve-roots goes far to explain this, but the cases

are too few and unfortunately but too rarely recorded in precise detail to admit of laying more weight on this point.

If "stiffness" be present it will be found to closely correspond with the portion of the tumour.

Similarly, if "curvature of the spine" is observed it will be found to be a secondary result of the tonic spasm of the spinous muscles, and therefore the concavity of the bend is on the same side as the growth. Removal of the source of the spasm, as in Case 32, restores the line of the spine.

#### *Column 17. The Pupils.*

As a symptom of this class of cases, change in the size and activity of the pupils only occurs when the cord is pressed upon above the level of the second dorsal nerve. When this happens, as in Case 4, the dilator fibres are of course paralysed, and the pupil becomes smaller than that of the opposite side, and almost immobile.

#### *Column 18. The Urine.*

As a rule the urine becomes retained in the bladder as the paraplegia is gradually developed, and following such retention comes decomposition and then cystitis. The organism (*Streptococcus pyogenes*?) which sets up the cystitis gains access by the ureters to the kidneys, and there develops, causing interstitial nephritis, the usual direct cause of death in these cases. In but four cases is the urine recorded as normal, viz. extradural scirrhus, extradural tubercle, intradural fibroma *not causing paralysis of any kind*, intradural sarcoma in which rapid generalisation occurred.

It is curious to note that in the first two instances the lower limbs were œdematous. No change would be expected in the third case.

This is not the place to enter into any discussion as to the cause of the alkalinity of the retained urine and its

subsequent decomposition. Suffice it to say that, as proved on p. 401, the reaction does not depend on the introduction of the *Micrococcus uræa* from without.

*In the scirrhus case polyuria occurred.*

*Column 19. Vaso-motor changes.*

The changes which may be attributed to perverted vaso-motor action are of course summed up in the word swelling. This was noted but six times in the whole fifty-eight, consequently it cannot be looked upon as an important symptom. Neither, unfortunately, can one learn from the accounts of the case why it should have occurred even thus rarely.

*Column 20. Vomiting*

Was never present as a direct result of the cord mischief.

The circumstances which relate to the tumour must now be grouped together for consideration.

*Column 21. Relation to the Dura Mater.*

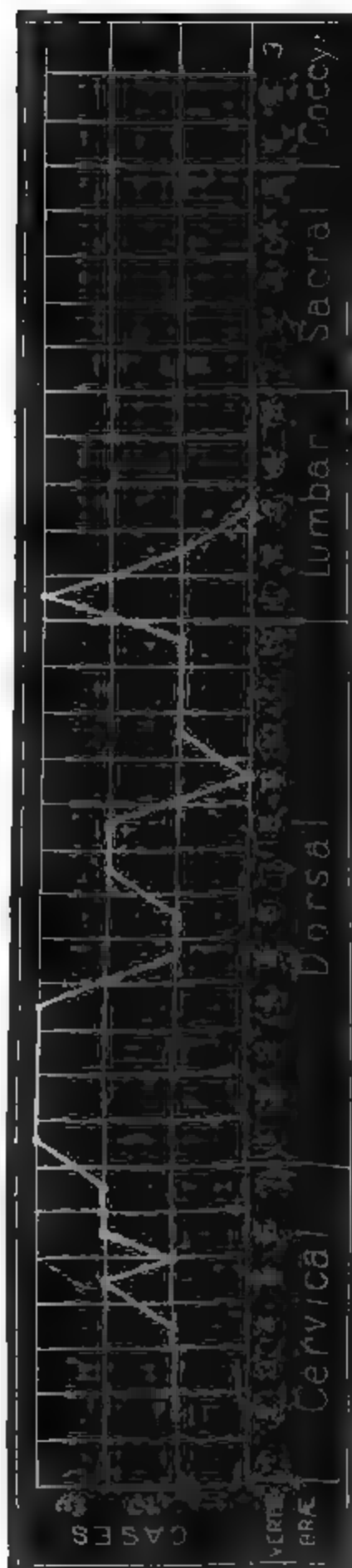
For every reason the relation of the growth to the dura is of the first importance. It has already been shown that we may thus separate tumours of different nature as being of practical certainty situated either inside or outside the theca. For the further bearings of this point on these cases reference must be made to the foregoing pages.

*Column 22. Size.*

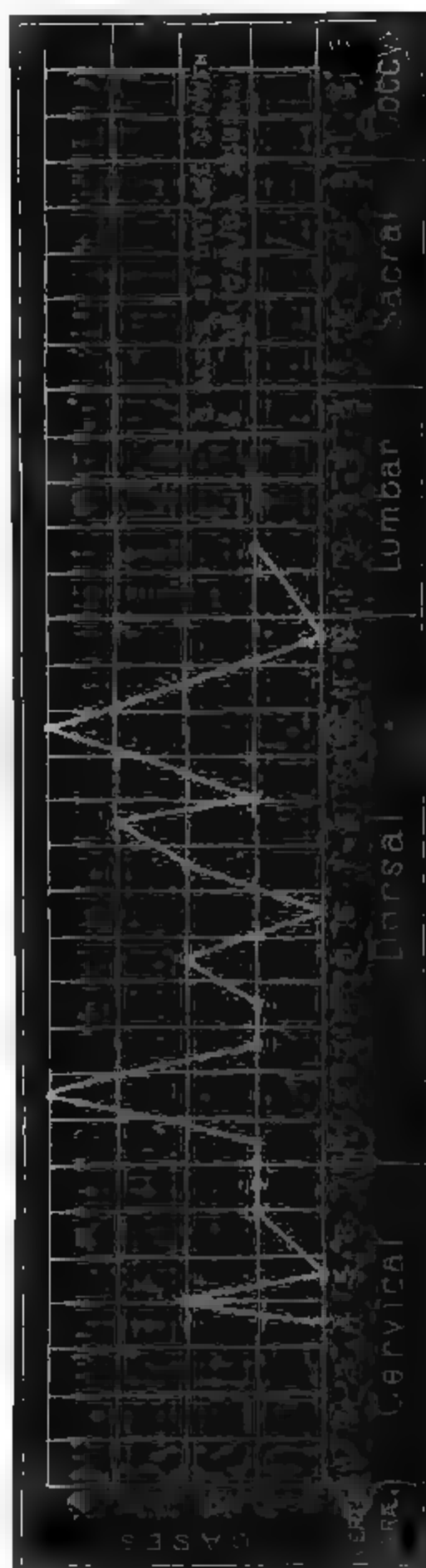
The size of the growth in any particular case is limited by the size of the theca, the amount of its lumen taken up by the cord, &c. As a rule the mass is oval or ellipsoidal,

*Curves showing the Prevalence of Growths at different Points in the Spine.*

*I.—Extradural.*



*II.—Intradural.*



*Note.*—The curve in the extradural group is necessarily of much less value than No. II in that in individual cases in the upper dorsal region affected so many vertebrae as to prevent localisation.

the long axis being parallel to the middle line of the cord. A necessary result of the variable position of the growth is that tumours of the same size produce widely different effects, consequently the size *per se* has little bearing on the symptoms.

### *Column 23. Position.*

There are several ways in which the position of the growth assumes importance, viz. :

1. In relation to the causation of the growth.
2. The relative effects of the growth. (a) According to its vertical position on the cord. (b) According to its horizontal position on the cord.

(a) On grouping all the cases in the Table together, it is at once obvious that most of the cases occur about the following points in the spine :

1. Just below the centre of the cervical region.
2. The upper end of the dorsal region.
3. The lower end of the dorsal region.

These regions are those in which there is least intrathecal spare space. Moreover, just as the cases increase in number from the cervical region downwards so the cord becomes less and less firmly fixed in the theca. Whether it is injudicious to regard all these facts as pointing to a traumatic<sup>1</sup> origin of the majority of the growths time alone will show. The cases in which there was good reason to suppose that direct injury was the exciting cause of the growth are not specially grouped around one of the above-mentioned regions, so that until we have before us the fuller and more accurate accounts of future cases we must leave undecided the bearings of the position of the growth.

It must be observed in passing that no one kind of growth is peculiar to any one region of the spinal column.

(b) The position of the growth in causing the production of definite symptoms, according as to whether it is on the anterior, posterior, or lateral columns, has already

<sup>1</sup> *E. g.* including direct injury, changes of temperature, &c.

been in great part described. But it remains to be mentioned that the *relative* rapidity of development of the pressure in these tumour cases seems to have prevented such minute analysis as is possible in pressure of the cord from caries of the spine producing curvature. Moreover, the diagnosis having usually been wrong, and attention having been concentrated on the pain, the initial stages have been so badly described as to make it impossible to place much trust on the accounts, as explanatory of the influence of position.

*Column 24. Nature of the Growth.*

The nature of the growths is set forth in the headings of each division of the table, and as far as possible the details of the structure of the tumour are supplied in each case. Leaving out the parasitic cysts, &c., the conclusion from the list of names is obvious, namely, that almost all these tumours are those of the simple connective-tissue type, and that they spring from the arachnoid, or more rarely, from the pia mater. With the debated origin of psammoma we have nothing to do here, but the epitheloid covering of the arachnoid sufficiently indicates its possible source. Although too, as before stated, some of the diagnoses of the real nature of the growths are uncertain, still, whatever the kind asserted, there is one characteristic common to all the localised intradural kind which is of the highest importance. This is the fact that they are almost invariably covered with a thin capsule (derived from the arachnoid?) and that their connection with the spinal cord is of the slightest. Consequently, they can be dissected from it without causing the slightest injury to a single nerve-fibre. In this connection reference may properly be made to the fact that since the tumours are as a rule of the simple connective-tissue type, extremely localised and slow growing (average duration of symptoms in intradural growths being about two and a half years, with the resul-

tant production of a small mass of tissue weighing a few drachms), recurrence after removal is not to be expected. In Case 32, the only one at our disposal for illustration of this point, there is not the slightest sign of recurrence one year after, nor is such probable.

*Column 25. Effect on the Cord.*

The effect on the cord varies widely, even among growths of the same nature, of much the same size, and apparently producing much the same effects, as far as general symptoms are concerned, and in cases where much the same kind of secondary changes appear to have occurred (see, for instance, Cases 47 and 48).

The cord was softened (*vide infra*) more frequently with intradural than extradural growths, the percentage for the former being 55, and for the latter 46, of the cases in which changes in the cord were noted as present or not. When softening occurs it appears always to pass through the stages of congestion (this being almost always noted as occurring in the neighbourhood of the tumour), red softening, yellow softening, and grey degeneration. All these require no detailed account. So too when the softening has interrupted any of the great tracts of fibres in the cord there ensues ascending and descending degeneration of the ordinary kind and extent.

One further point with regard to the softening requires notice, and that is its distribution. Of course naturally it is most marked opposite the centre of the growth, but its farther extension is of course a matter of much importance and interest. Of six cases in which it is recorded to have occurred, in four it spread downwards and in two upwards, one very slightly. Doubtless this distribution is due to thrombosis of the great median medullary vessels.

*Column 26. Treatment.*

For all the horrible sufferings of the fifty-eight cases in the Table, in only two was any treatment of avail, viz. Nos. 4 and 32. In the former case excision of a part of the growth relieved the pressure, and so the symptoms for a time. In the second the complete removal of the growth has, it may be hoped, obtained permanent relief. Unfortunately the condition of the patient, owing to the errors of diagnosis, has usually been made more hopelessly miserable by free use of the actual cautery, moxas and blisters, while in other instances the additional employment of mercury, iodide of potassium, &c., has been resorted to.

*Column 27. Records of the Autopsy, especially with respect to the Presence or Absence of any other Lesion which, independently of the Tumour, would have caused Death.*

The melancholy inspired by consideration of Column 26 is intensified by the facts of Column 27, for in no less than 74 per cent. of the extradural growths, and 83 per cent. of the intradural, the patient died simply from the direct effects of the tumour, *i. e.* from exhaustion (in a very large number of cases), owing to pain, &c., or from pyæmia owing to absorption from the bedsores, or from septic pneumonia, or from acute septic interstitial nephritis.

Roughly speaking, therefore, about 80 per cent. of these miserable cases could have been relieved entirely by operation, and those which were hopeless might by relief of pressure have been granted a euthanasia.

The simple effects of confinement and nerve exhaustion are seen in the four instances in which fatty degeneration is noted among the effects of the intradural growths.



*Conclusions.*

The lessons of the facts detailed in the foregoing pages are so extremely obvious that very few words are required to set them forth. They amount to this, that, granted the diagnosis is correctly made, there is but one treatment, viz. removal of the source of pressure by operation. It may at once be said, and rightly, that the question of diagnosis in the large majority of cases arises when only as yet one cardinal symptom is before us, most commonly pain, and that therefore a diagnosis of such certainty as to warrant exploration is not possible. To this nothing can be objected in view of the responsibility the surgeon takes upon himself, but at least absolution from the major part of such responsibility is obtained with the discovery of the first localising symptom independent of the constancy, the position, and the character of the pain. The differential diagnosis of the cause of painful paraplegias, material for which is given in the table, considered fully, would be quite beyond the object of the present paper, and must be reserved for another occasion. A close survey of the conditions under which tumours have been found will meanwhile afford the best aid to the recognition of the real nature of doubtful cases.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' New Series, vol. ii, p. 407.)



### **DESCRIPTION OF PLATE III.**

**A Case of Tumour of the Spinal Cord (W. R. GOWERS, M.D., F.R.S.,  
and VICTOR HORSLEY, B.S., F.R.S.).**

A photograph was taken about July 31st which gives a view of the back of the patient, showing the scar of the operation. The spots around the scar are traces of small pustules, which, however, gave the patient practically no trouble, and which were caused no doubt by the irritation of the large quantity of cerebro-spinal fluid soaking the dressings and dissolving the perchloride of mercury, &c., contained therein.

PLATE III.

MED. CHIR. TRANS. VOL. LXXI.





FROM A PHOTOGRAPH.



meter ages.	Vomiting.	a. R. to
9	20	1
	—	Out di
	—	Out di
	—	Ext di
	—	(1) O the mov cess (2) O the the and
	—	Out di
	—	Out di
son of his	Frequent and severe (suffered much from bronchitis)	Out di
	—	Out di

No of case		Sex, age.	Alleged cause.	History.				
1	2	3	4	5	6	7	8	9
			Personal.	Family.	a. Total duration.	b. Duration under observation.	c. Location.	d. Duration.
<b>Sarcoma (continued) :</b>								
9	M. 10	A fall on the back	—	—	—	—	to 8th month of (3) paraplegia of the lower limbs	to 8th month of (3) paraplegia of the lower limbs
<b>Tubercle :</b>								
10	M. 36	—	—	—	—	—	back, and, late loss of power & ability in the lower limbs; a corn in the back was	to 4th month of paraplegia of the lower limbs
11	F. 60	None known.	—	—	3½ years	3 years	Throats, the lower limbs, the	



No.	Sex.	Age.	d. Nature.	e. Effect on cord.	Treatment.	Result.	Autopsy notes of other fatal lesion.	Reference.
			24	25	26	27	28	29
1	Male	14	Two echinococcus cysts, containing daughter cysts	Excess of cerebro-spinal fluid. Cyst No. 1 flattened, for 3.5 cm. by 1.8 cm., the cord on the posterior surface of the left side, so that towards the upper border of the depression the cord was almost divided, there being only a flattened strip of cord left a few mm. broad, and very soft. Vessels in neighbourhood of both cysts very vascular; very empty at points compressed. Cyst No. 2 also compressed the cord, and rendered it soft	—	Death	Caseous mass in apex of left lung, oedema of lung, bronchitis, pyelitis, cystitis	Bartels, Kiel Deutsches Archiv für klinische Medizin, Bd. v, 1869, p. 108.
2	Male	14	Cysticercus	Appeared softened; doubtful, as decomposed	Blisters, Pot. Brom., arsenic, &c.	Death	Cysticerci in brain; nil else important	Westphal, Berliner klinische Wochenschrift, No. 43, 1865, p. 425.
3	Male	14	Hard, opaque, yellow, amorphous	—	—	Death	Few gummata in liver and lungs	Wilks, Guy's Hosp. Reports, 3rd series, vol. ix, p. 60, 1868.
4	Male	14	Not stated	Spinal cord was slightly flattened by the growth, but otherwise quite healthy	—	Death	Nothing noted	Leyden, Klinik der Rückenmarks Krankheiten, Band i, 1874, p. 468.

## INDEX.

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*These Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted, as soon as printed, in the Library copy, where the entire Index to the current date may always be consulted.*

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